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CONTENTS OF PREVIOUS NUMBER

FEBRUARY 1948. NUMBER 2

Degeneration and Regeneration of Nerves in Corneal Transplantation: An Experimental Study. Humberto Escapini, M.D., New York.

Tests of Defective Color Vision: Reflections Based on Experience in Testing Persons with Defective Color Sense. Burton Chance, M.D., Philadelphia.

Iridotasis ab Externa for Relief of Glaucoma, Simplex. William A. Stoutenborough, M.D., Columbus, Ohio.

A Tangent Screen Illuminator: Construction, Use and Advantages. Conrad Berens, M.D., New York, and Matthew Luckiesh, D.Sc., Cleveland.

Ambylopia ex Anopsia: A New Concept of Its Mechanism and Treatment. B. M. Lazich, M.D., Los Angeles.

Congenital Impatency of the Nasolacrimal Duct. DuPont Guerry III, M.D., and Edwin L. Kendig Jr., M.D., Richmond, Va.

Spontaneous Absorption of Congenital Cataract Following Maternal Rubella. Leon H. Ehrlich, M.D., New York.

Distance Discrimination. Monroe J. Hirsch, Ph.D., Columbus, Ohio, and Frank W. Weymouth, Ph.D., Stanford University, Calif.:

I. Theoretic Considerations.

II. Effect on Threshold of Lateral Separation of the Test Objects.

Sodium Sulfacetimide in Ophthalomology. Leo L. Mayer, M.D., St. Louis.

Obituaries:

Ervin Török, M.D.

News and Notes.

Abstracts from Current Literature.

Book Reviews.

ARCHIVES OF OPHTHALMOLOGY

VOLUME 39 (old series Volume 96) MARCH 1948 NUMBER 3

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EXPERIMENTAL STUDIES OF OCULAR TUBERCULOSIS

IX. Failure of Penicillin to Affect the Course of Experimental Ocular Tuberculosis

JOHN D. KENNEDY, M.D.

ALAN C. WOODS, M.D.

JOHN BUCKLEY, M.D.

MORGAN BERTHRONG, M.D.

AND

EARL L. BURKY, M.D.

BALTIMORE

THERE is little in the literature concerning the effect of penicillin on Mycobacterium tuberculosis. Abraham, Chain and Florey,[1] as a result of their in vitro experiments, reported the tubercle bacillus to be insensitive to penicillin. In Woodruff and Foster's experiment,[2] tubercle bacilli (type and virulence unstated) were not inhibited by 1,000 units of penicillin per cubic centimeter of culture medium. They attributed the insensitivity of the tubercle bacillus to the fact that it is among the most active destroyers of penicillin. Smith and Emmart [3] found that neither a concentration of 3,000 units of penicillin per hundred cubic centimeters of culture medium prevented growth of a virulent human type of bacillus in vitro nor did 800 units of penicillin affect significantly the degree of tubercle development in the chorioallantoic membrane. Gerber and Gross [4] reported that a concentration of 1,500 units per cubic centimeter of culture medium did not inhibit a rapidly growing nonpathogenic strain of tubercle bacilli. Only two reports are available on the effect of penicillin on the disease in experimental ani-

This work was supported by a grant-in-aid from the John and Mary R. Markle Foundation.

From the Wilmer Ophthalmological Institute and the Department of Pathology of the Johns Hopkins Hospital and University.

1. Abraham, E. P.; Chain, E.; Fletcher, C. M.; Gardner, A. D.; Heatley, N. G.; Jennings, A. B., and Florey, H. W.: Further Observations on Penicillin. Lancet **2**:177 (Aug 16) 1941.

2. Woodruff, H. B., and Foster, J. W.: In Vitro Inhibition of Mycobacteria by Streptothricin, Proc. Soc. Exper. Biol. & Med. **57**:88 (Oct.) 1944.

3. Smith, M. I., and Emmart, E. W.: The Action of Penicillium Extracts in Experimental Tuberculosis, Pub. Health Rep. **59**:417 (March 31) 1944.

4. Gerber, I. E., and Gross, M.: Inhibition of Growth of Mycobacterium Tuberculosis by a Mold Product, Science **101**:616 (June 15) 1945.

mals. Robinson [5] carried out two series of experiments, in each instance infecting mice with an avian strain of Myco. tuberculosis. In the first experiment, the organisms were suspended in a 1.6 per cent solution of penicillin for forty-eight hours and then injected in varying doses into mice, which were killed after three or four weeks. In the second experiment, the infected mice were treated with a dose of 1,000 units of penicillin per mouse per day for three weeks. No effect on the course of the infection was noted in either experiment. The only animal experiment in which a mammalian bacillus was used is that of Smith and Emmart.[3] They infected guinea pigs with a human type of bacillus. There were 13 treated animals and 16 controls in their series. The treated animals were given 500 units of penicillin daily for the first four days and then 200 units daily for the next twelve days. The dose was reduced because 4 animals died of drug toxicity during the first few days of treatment. Penicillin appears to be remarkably toxic for the guinea pig (Hamre and associates [6]). Inadequate supplies of penicillin caused the treatment to be discontinued after only sixteen days. At the end of forty-five days the experiment was terminated. No beneficial effect of the penicillin treatment was noted.

The statement that penicillin has no effect on the tubercle bacillus or on the course of the disease has gained universal credence. The published reports of the in vitro tests do not indicate any degree of sensitivity under these conditions. The two experiments carried out on experimental animals are inconclusive, owing to the use of an avian strain and an unsuitable animal in one [5] of the experiments and of an inadequate dose over too short a period of treatment in the other.[3] The question is of such importance that it was thought worthy of reinvestigation with larger doses of the drug, more particularly since, with the use of a massive dose of penicillin, bacterial endocarditis and syphilis, originally thought insusceptible to the drug, are now known to respond favorably. Furthermore, the work of Harper and Blain [7] and of Blain and Kennedy,[8] in which a massive dose of penicillin prevented invasion of the damaged intestinal wall in dogs by the gram-negative flora, lent encouragement to the hope that tubercle bacilli, also rated as insensitive to penicillin, might be amenable to similar treatment.

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5. Robinson, H. J.: Toxicity and Efficacy of Penicillin, J. Pharmacol. & Exper. Therap. **77**:70 (Jan.) 1943.

6. Hamre, D. M.; Rake, G.; McKee, C. M., and MacPhillamy, H. B.: The Toxicity of Penicillin as Prepared for Clinical Use, Am. J. M. Sc. **206**:642 (Nov.) 1943.

7. Harper, W. H., and Blain, A., III: The Effect of Penicillin in Experimental Intestinal Obstruction: Preliminary Report on Closed Loop Studies, Bull. Johns Hopkins Hosp. **76**:221 (June) 1945.

8. Blain, A., III, and Kennedy, J. D.: The Effect of Penicillin in Experimental Intestinal Obstruction: Studies on Strangulated Low Ileal Obstruction, Bull. Johns Hopkins Hosp. **79**:1 (July) 1946.

In the experiments here reported the rabbit eye was chosen as the site of the tuberculous lesion to be studied. The course of ocular tuberculosis resulting from inoculation of the anterior chamber of the normal rabbit eye with the human type of tubercle bacilli is well known. The resulting ocular disease in such normal rabbits lasts about three months before final perforation or healing. The animals rarely die of concurrent systemic tuberculosis. Accurate clinical observation and evaluation of the course of the disease, as well as final histologic estimation, are possible. The distribution of penicillin in the ocular tissues after systemic injection is comparable to that found in other body tissues, smaller amounts occurring in the ocular fluids. Von Sallmann and Meyer [9] showed that thirty minutes after the intramuscular injection of 30 mg. of sodium penicillin (the equivalent of 2,000 to 3,000 Oxford units) into rabbits the aqueous contained 0.69 microgram per cubic centimeter and had moderate antibiotic action. Struble and Bellows [10] gave dogs one large intravenous dose (12,800 units of penicillin per kilogram of body weight) and removed the eyes at varying intervals. The penicillin content of the various ocular tissues and fluids was assayed by the Florey method. Penicillin was detected in the eyeball within fifteen minutes. At the end of one hour the concentration was between 10 and 15 units per gram in the sclera, 1.39 units per gram in the chorioretinal layer and approximately 1 unit per cubic centimeter in the aqueous. Thereafter the values declined and barely a trace of penicillin remained in the eye after three hours. Town and Hunt [11] found in 35 rabbits that an intramuscular dose of 7,000 units per kilogram of body weight gave a therapeutic level of penicillin in the aqueous sixty minutes after injection, the loss thereafter being gradual.

EXPERIMENTAL METHODS

Sixty-five normal rabbits were inoculated in the anterior chamber of the left eye with 0.2 cc. of a paper filtrate of a six week old culture of a virulent human type of tubercle bacilli. The inoculum was diluted to contain an average of 12 organisms per oil immersion field. Preliminary experiments showed that this dose produced slowly progressive, satisfactory ocular tuberculosis in the normal rabbit. The eyes were examined daily for the first three days after inoculation and thereafter at weekly intervals. The intensity of the ocular tuberculosis—ciliary congestion, tubercles and infiltration of the cornea, tubercles and inflammation of the iris, opacities of the vitreous, lesions of the fundus, caseation and rupture—was evaluated on a numerical scale from ¼ to 4 in each eye, the final figure representing widespread caseation with imminent or actual rupture of the

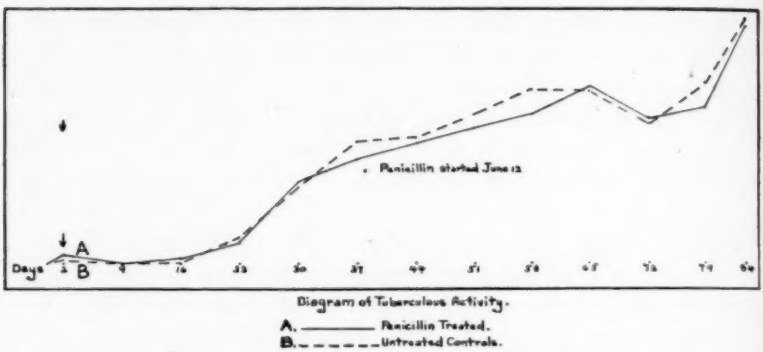
9. von Sallmann, L., and Meyer, K.: Penetration of Penicillin into the Eye, Arch. Ophth. **31**:1 (Jan.) 1944.

10. Struble, G. C., and Bellows, J. G.: Studies on the Distribution of Penicillin in the Eye and Its Clinical Application, J.A.M.A. **125**:685 (July 8) 1944.

11. Town, A. E., and Hunt, M. E.: Concentration of Penicillin in the Aqueous Humor Following Systemic Administration, Am. J. Ophth. **29**:171 (Feb.) 1946.

eye. Five and one-half weeks after inoculation, 32 rabbits were chosen with early, but definite, lesions, with an average rating of about 1.0 on the numerical scale, and were divided into two groups on the basis of equal severity. The animals in the first group, the severity of whose lesions was rated as 1.3 on the numerical scale, were then treated with penicillin, while the second group, with a rating of 1.2, were left untreated as controls.

Each treated rabbit received 200,000 units of penicillin per day.[12] Individual doses of 50,000 units were administered each day by subcutaneous injection at 8 a.m., 1 p.m., 7 p.m. and 12 midnight. The duration of treatment was forty-five days. On the basis of body weight this dose for a 2.5 Kg. rabbit would be the equivalent of 5,200,000 units per day for a man weighing 65 Kg. The following levels in the blood were obtained on 2 rabbits. For rabbit 69, the blood level just before injection was less than 0.025 Oxford unit per cubic centimeter; after fifteen minutes it was 8.35 units; after thirty minutes, 13.36 units; after one hour, 11.9



Tuberculous activity in the eyes of rabbits treated with penicillin (curve *A*) and of untreated (control) rabbits (curve *B*). Arrows indicate inoculation with tubercle bacilli of the anterior chamber of the left eye on May 11, 1946.

units; after two hours, 2.67 units, and after four hours 0.26 unit, per cubic centimeter. For rabbit 97, the levels for the corresponding times were 0.025 unit, 13.36 units, 12.19 units, 5.5 units, 2.0 units and 0.2 unit per cubic centimeter.

RESULTS

Clinical Results.—The daily treatment was maintained for a period of forty-five days. The eyes were routinely examined at least once a week, and the degree of the tuberculous lesions was evaluated. The course of the disease in the treated and in the untreated group is shown on the figure, curve *A* representing the course in the penicillin-treated rabbits and curve *B* the course in the untreated controls. As shown by the two curves, the course was practically identical in the two series, there being no clinical evidence of any inhibitory effect on the ocular

12. The penicillin was supplied through the courtesy of Merck & Co., Inc., Rahway, N. J. This particular product was calcium penicillin G, containing a small amount of unknown impurities. The potency, as measured, represented penicillin G alone.

tuberculosis in the treated group. The course of the tuberculosis in these eyes was the same as that usually observed in the normal rabbit. Three rabbits of the treated group and 2 rabbits of the control group died in the course of the experiment, leaving 13 treated animals and 14 controls for evaluation of final results. At that time, of the treated animals, the eye had ruptured in 3 and was about to rupture in another. The average severity of the lesions on the numerical scale was 2.9. In the control group, 7 of the diseased eyes had ruptured, and the average severity of the lesions was 3.0. In 3 rabbits of the control series and in 1 rabbit of the penicillin-treated series the disease appeared to be healing spontaneously.

Pathologic Observations.—The animals were killed and autopsied on the forty-fifth day of treatment. The infected eyes were removed and histologic preparations made. The lens was removed, and equatorial

Measurements of Areas of Tuberculosis in Rabbits Treated with Penicillin and in Untreated Rabbits

		Areas of Tuberculosis (Sq. Mm.) in Microscopic Sections					
		Eye			Viscera		Total Area of Tuberculosis (Eyes and Viscera)
Group	Clinical Activity Scale 1/4-/4	Non-caseous	Caseous	Total Tuberculosis for Eye	Non-caseous Tuberculosis	Caseous Tuberculosis	
Untreated controls (14)							
Totals	41	71	63	134	9	7	150
Averages	3	5	4.5	9.5	0.6	0.5	10.6
Penicillin-treated rabbits (13)							
Totals	37	41	30	71	11	0	82
Averages	2.9	3.2	2.3	5.5	0.8	0	6.3

sections containing all the other structures of the eye were made. No macroscopic lesions of the viscera were seen at autopsy. Sections for microscopic examination were made of the lung, liver, spleen and kidneys of each animal, in addition to those of the eye. Microscopic lesions of the viscera were observed in 6 of the treated rabbits and in 6 of the controls also.

Comparison of the degree of tuberculous involvement on a numerical basis was attempted by measuring separately the areas of non-caseous and of caseous tuberculosis, using an eyepiece fitted with a squared micrometer disk and a 32 mm. objective. Doubtful lesions were checked with a 16 mm. objective. There were less total ocular tuberculosis, less total ocular caseation and less total tuberculosis, both ocular and visceral, in the penicillin-treated group. There was a wide overlap, however, in the individual animals in each group. The total and average measurements for the two groups are given in the table. A statistical analysis revealed that the probability that chance alone would produce differences of such a size was just over 20 per cent. Under the conditions

of the experiment, the fact that less tuberculosis was observed in the microscopic sections from the penicillin-treated rabbits is not statistically significant. In view of the negative results, detailed figures for the individual animals are not presented.

COMMENT

The observations reported here require little comment. Penicillin G, given in massive doses, had no appreciable chemical effect on the course of inoculation ocular tuberculosis in the nonimmune rabbit. It likewise had no effect in preventing the spread of the organisms from the eye to the viscera, 6 animals in each series showing microscopic visceral lesions. The only possible deterrent action which could be attributed to the penicillin is the numerically lower degree of noncaseous and caseous tuberculosis in the treated group as compared with the control group. However, in each group there was a wide variation in the individual rabbits, and an analysis of these figures showed them to be without statistical significance. The conclusion that this particular sample of penicillin G had no therapeutic effect on ocular tuberculosis in the nonimmune rabbit is inescapable.

CONCLUSIONS

Large doses of calcium penicillin G (200,000 units per day, equivalent in amount to 5,200,000 units for a man weighing 65 Kg.) had no significant effect on experimental ocular tuberculosis in the nonimmune rabbit.

Dr. Arnold R. Rich gave valuable aid in this study, and Dr. Margaret Merrell, lecturer in preventive medicine, made the statistical analysis.

SPASM OF MACULAR ARTERIES

Report of a Case

SIE-BOEN-LIAN, M.D.
BATAVIA, JAVA

IN HIS papers of 1929 and 1937, Hörniker [1] stressed that the terminal macular vessels are easily injured by disturbances, especially of an angioneurotic (angiospastic) kind. The case of spasm of the macular arteries reported here presented a different picture from that of many macular disturbances attributed by Hörniker to spasm of the terminal vessels, but the analogy is clear.

The case was one of obstruction which within a few days affected nearly all the macular arteries, including the cilioretinal artery. I had the opportunity of treating the patient for more than a year. In the beginning, I examined him daily; later, examinations were made at longer intervals. During the daily examinations of the fundus, I saw interesting changes in the affected vessels which seemed to me worthy of record.

REPORT OF CASE

History.—O. H. T., a Chinese aged 34, presented himself on Aug. 25, 1944, with the complaint of blurring in his right eye the day before. Examination revealed normal visual acuity in both eyes. There were no changes in the fundus. On September 10 he consulted me again because of poor vision in the right eye for two days. After his first visit he had had a few attacks of blurred vision in the right eye, usually lasting half an hour. During the past week he had had short attacks of weakness of the left arm with cutaneous paresthesia of that arm and of the left side of the head. There was no migraine or syphilis at the time of the examination. General examination revealed a healthy appearance with no evidence of abnormality of the heart.

Ophthalmologic Examination.—September 10: Right eye: Vision was limited to counting fingers. The cornea and media were clear. There was slight pallor of the lower half of the macular region except in an area of ½ disk diameter around the fovea centralis. This region is supplied by three branches of the inferior temporal artery. The pallor extended below to the second macular branch, nasally to the disk and temporally to the terminal part of the third macular branch, about 1 disk diameter to the temporal side of the fovea centralis. The last half of the second macular branch had been converted into a thin white line. The first macular branch was rather thin. The third macular branch was also somewhat narrowed and showed the following changes: Beginning just after this branch crosses the inferior temporal vein, and extending distally beyond the bifurcation for a distance of nearly 1 disk diameter, the vessel was converted

1. Hörniker, E.: Klin. Monatsbl. f. Augenh. **98**:487, 1937.

into a white column, which was sharply demarcated from the red background. Beyond this white column both vascular branches were beaded. Some of the ends of the affected artery had changed into thin white lines. The corresponding vein showed beading. There was a paracentral scotoma (fig. 1).

Left eye: The eye was white; the cornea and media were clear. The fundus was normal. Vision was 5/4.

Treatment: Intravenous injections of sodium nitrite were given.

September 11: The changes in the inferior macular branch had disappeared. The caliber of the vessels had somewhat increased. The pallor of the lower half of the central portion of the fundus persisted. The cilioretinal artery was more contracted and appeared white for a longer distance. Edema of the upper half of the macular area caused pallor of the whole central portion of the fundus except for the foveal region. In two places the terminal portions of the small upper branches of the macular arteries looked like white lines.

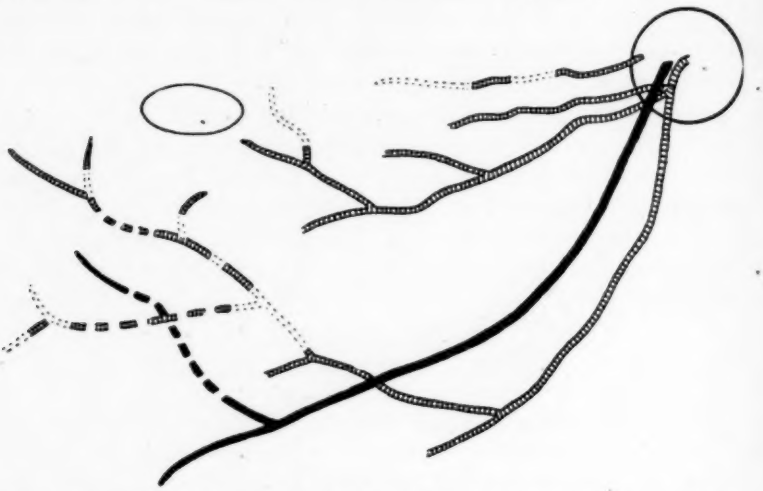


Fig. 1.—Spasm of the macular arteries in the right eye (Sept. 10, 1944).

September 12: The macular vessels showed no abnormality except for the cilioretinal artery and the first inferior macular branch. Edema in the lower half of the central portion of the fundus was decreasing.

September 14: The terminals of the middle small branch of the superior macular artery and the distal part of the superior temporal artery were converted into white lines, with red spots in the former. There was beading of the superior macular vein, which crossed the affected artery.

September 15: The beading of the macular vein had completely disappeared. The affected part of the superior temporal artery showed small red segments in some places in the white column.

September 16-27: Daily examination of the fundus showed constant changes in the appearance of the affected area. Some of the small arterial branches, which were white formerly, had become red again or showed red particles in the white lines. On the other hand, new small vessels had become converted into white lines. The central area of the fundus, supplied by these vessels, became opaque after the vessels had become affected. Moreover, the white segment in the arteries changed constantly in size, the demarcation between the white and the red area moving

centrally or distally. On September 15, just after injection of 100 mg. of sodium nitrite, movement for a distance of 2 disk diameters in the direction of the disk was noted in the demarcation between the white and the red area in the peripheral part of the superior temporal artery. There was no progressive movement in the affected arteries; the demarcation between the white segment and the normal red blood column of the arteries moved to and fro for a very small distance with the cardiac systole. This rhythmic movement could be seen not only in the main branch but also at places far out in the periphery.

When pressure was applied to the globe, the white area in the distal part of the superior temporal artery moved back centrally, but did not pass a bifurcation. When the pressure was released, the white column returned to its former place, without invading the other branch of the bifurcating artery. This movement of the white column with pressure on the globe did not take place at the extreme ends of the arterial branch.

September 19: The affected end of the superior temporal artery proximal to the bifurcation showed an area of constriction of about 1 disk diameter, but then became almost normal in size (fig. 2).

September 20: Immediately after injection of 100 mg. of sodium nitrite intravenously, examination of the fundus showed a striking picture. The main

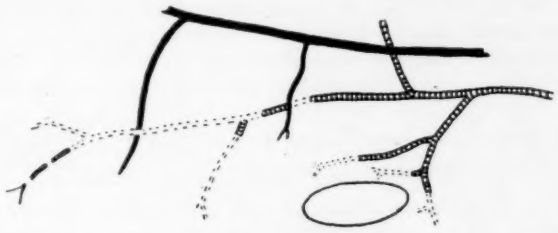


Fig. 2.—Constriction of the superior temporal artery (Sept. 19).

superior and inferior arterial branches suddenly became pale. The change from red to white extended from the periphery, progressing rapidly to the disk. The caliber of the affected arteries remained normal. For a few seconds there was no circulation in the two arteries except for the to and fro movement of the demarcation between the red and the white area with the cardiac systole; then a slow movement of the blood column toward the white area took place. This blood column was somewhat beaded at the end. With each systole it moved forward until the whole vessel became red again. In addition, it was noted that the white area in the peripheral part of the superior temporal artery extended toward the disk. After one-half hour the white column had not yet reached its position before the injection. Vision in the right eye was 2/60.

September 21: No change was apparent. Up to this time no new arteries had been affected. Slowly, the white part of the affected arteries became red again, and the area supplied by these vessels slowly regained its normal red color. Acuity of vision in the right eye improved slowly.

October 10: No new beading was to be seen, and there were no white lines at the ends of the small arterial branches. Vision in the right eye was 5/30 partly. The peripheral part of the superior temporal artery and the cilioretinal artery were smaller than normal. The field was unchanged. There was a slight opacity along the cilioretinal artery. The normal foveal reflex was not noted. The macular area appeared somewhat granular, with here and there white glistening spots.

Feb. 23, 1945: On the formerly affected parts of the retina a few glistening points could be seen.

June 23, 1945: The upper macular branches were still thin. Vision in the right eye was 5/30 partly.

September 15: The patient was admitted to the hospital because of a tuberculous process in the apexes of both lungs.

Oct. 3, 1946: There were no changes in the ocular condition, and no attacks of weakness of the arms or paresthesia had occurred.

Summary.—The case is that of a man without cardiac disease who one year after the first ocular examination presented slight tuberculous involvement of the lungs. The ocular disturbance was preceded by attacks of transient blurring, weakness of the left arm and cutaneous paresthesia of the left side of the head. The disturbance affected the macular region of the right eye, which was pale, first, in the lower half and, later, in the upper half. The affected vessels presented the following changes: Most of the terminal parts of the macular arteries and the cilioretinal artery were converted into fine white lines. The peripheral parts of the branches of the inferior and superior temporal arteries showed white areas, followed by segmentation of the blood column. The appearance of the macular vessels changed constantly. The inferior temporal artery was normal again on the second day of examination. The alteration in the superior temporal artery lasted about two weeks. On one occasion, a sudden change of two arteries into white columns was noticed during an ophthalmologic examination. Within a few seconds the affected vessels became normal again.

INTERPRETATION AND ANALYSIS OF OPHTHALMOSCOPIC OBSERVATIONS ON VASCULAR CHANGES

The white lines in the terminal parts of the macular arteries undoubtedly represented spastic empty vessels. The same phenomenon appeared in Grimsdale's [2] case of spasm of a branch of the central artery. These changes cannot represent small columns of plasma, for when pressure was applied to the eyeball no movement in the demarcation between the white line and the red blood segment was observed. As the white lines became red again within a shorter or longer period, they cannot represent obliterated ends of arteries. The white area in the more central part of the affected arteries, I am convinced, was of the same nature as the white segments in the beaded blood column, and was also a column of plasma. The phenomenon has nothing to do with embolism, in spite of the similarity of the white areas to emboli, which Coverdale [3] diagrammatically represented in his paper (fig. 3).

The changes in the retinal artery in Benson's case of spasm of a branch of the central retinal artery have much similarity to the alteration of the arteries in my case. In discussing Benson's case, Leber [4] suggested that the white part of the affected artery was simply a part of the vessel, filled only with plasma. In the present case, the white part of the artery was not constant in size; it began at the periphery and extended to the center. This fact supports the view that it was not an embolus. It was also of the same nature as the white column, into which a whole main arterial branch could be changed.

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2. Grimsdale, H.: Brit. J. Ophth. **24**:208, 1940.

3. Coverdale, H. V.: Brit. J. Ophth. **13**:529, 1929.

4. Leber, T., in Graefe, A., and Saemisch, T.: Handbuch der gesamten Augenheilkunde, Leipzig, Wilhelm Engelmann, 1911, vol. 7.

The change of the two main branches of the central artery into white columns and their restoration, observed during the short period of an ophthalmoscopic examination, has, so far as I have ascertained, never before been published. It may be regarded as an accelerated development of the process of alteration in other vessels. The whole process, which usually lasts many days, took less than one minute. This observation suggests a vasomotor reaction. A change in the vessels which passes off within such a short time can have only a vasomotor origin. As there was no narrowing of the arteries, the possible spasm did not involve the central part of the vessels, but affected only the terminal parts and the capillaries. In these arteries in which the end branches were obstructed by spasm the circulation of the blood stopped. Aggregation of the red cells occurred, accounting for the presence of columns consisting only of plasma. The injection of sodium nitrite had nothing to do with the vascular change; it was only coincidental that this change took place just after the injection. After each of more than twenty injections I examined the fundus, but only twice did I see changes in the vessels. There was no circulation in the affected arteries; with each cardiac systole the demarcation between the red and the white segment moved forward a short distance, but the next moment it returned to its former place. This to and fro movement was seen far into the periphery of the arteries, indicating that the vessel was not obstructed up to that peripheral point, and that the obstruction must be looked for beyond.

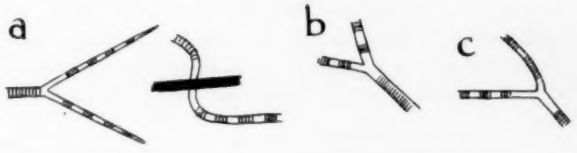

Fig. 3.—Segmented appearance of the central retinal artery (*a*) in Coverdale's case, (*b*) Butler's [4] case and (*c*) case presented.

DIAGNOSIS

The clinical appearance suggests an obstruction of the macular arteries. Spasm of the arterial wall is the most probable cause of the obstruction. The diagnosis of spasm is based on the following observations: (1) prodromal obscurations of vision; (2) absence of a cardiac lesion; (3) occurrence of attacks of weakness of the arm and paresthesias, pointing to spastic narrowing of certain cerebral vessels; (4) occasional alteration in the caliber of the affected vessels and changes in the terminal arterial branches into thin white lines, and (5) simultaneous involvement of the macular branches of the central retinal artery and of the cilioretinal artery.

The appearance of the changes in the vessels in cases of retinal disturbance recorded in the literature as being due to spasm of the vessels is not wholly similar to that in the present case. Many authors observed constriction of all the arteries (Halbertsma,[5] Nasir Farid Bey,[6] Maria Rosenstein [7]) with normal (Nasir Farid Bey [6]) or contracted (Rosenstein [7]) veins. Halbersma [5] noted beading of the veins. Hairi [8] saw a change of the inferior temporal artery in a white

5. Halbertsma, K. T. G.: Am. d'ocul. **168**:641, 1926.
6. Bey, N. F.: Brit. J. Ophth. **14**:402, 1930.
7. Rosenstein, M.: Klin. Monatsbl. f. Augenh. **75**:257, 1925.
8. Hairi, H.: Ann. d'ocul. **158**:662, 1926.

line, and Harms noted such changes in all retinal arteries. Kraupa and Hahn,[9] in a case of sudden blindness in a 5 year old child, noted edema of the retina, with normal arteries but enlarged veins, and attributed this disturbance to spasm of the capillaries. This view was strongly opposed by Kubik.[10]

Enlargement of the retinal veins was seen by Feigenbaum [11] in a case of transitory blurring of vision. The arteries were normal, and Feigenbaum suggested that the obscuration was due to obstruction of the venous return and that this might be caused by retrobulbar spasm of the central vein.

As already noted, there is evidence that the spasm occurs in the terminal parts of the arterial branches or in the capillaries. This spastic contraction may be an allergic vascular reaction. In the macular region, the choroidal capillaries show the densest reticular network, contain the largest quantity of blood and have the slowest circulation. These factors favor the accumulation of the antigens circulating in the blood (Behr [12]). Organisms of low virulence, moving from a distant focus to the macular region, may produce antibodies in this area. A new mass of the micro-organisms from the focus encounters the allergic tissue; antibody and antigen come into association, with release of a toxic substance of a histamine nature, which has an angiospastic effect.

In the present case the observation of a mildly tuberculous process in the lung a year after the first examination points to the existence of a latent tuberculous focus somewhere in the body at the time of onset of the ocular disease, with production of an allergic state.

SUMMARY

A case of obstruction of the macular arteries, including the cilioretinal artery, is described. The obstruction is diagnosed as spasm of the ends of the affected vessels, probably of allergic origin. The allergic state is thought to be due to the tubercle bacillus.

9. Kraupa, E., and Hahn, L.: Klin. Monatsbl. f. Augenh. **66**:829, 1921.
10. Kubik, F.: Klin. Monatsbl. f. Augenh. **68**:366, 1922.
11. Feigenbaum: Klin. Monatsbl. f. Augenh. **48**:190, 1922.
12. Behr, cited by Zeeman, W. P. C.: Geneesk. bl. u. klin. en lab. v. d. prakt. **41**:69, 1946.

PATHOGENESIS OF MYOPIA

A New Classification

FREDERICK C. STANSBURY, M.D.
NEW YORK

PRIOR to von Helmholtz' epochal invention of the ophthalmoscope in 1851, little was established concerning the pathology of myopia. Kepler,[1] early in the seventeenth century, expressed the belief that in accommodation the retina moved closer to the lens and that myopia was a disturbance of this function. Plempius,[2] in 1632, first examined the myopic eye anatomically and noted the increased distance between the lens and the retina. Boerhave,[3] in 1708, confirmed the findings of Plempius and suggested this axial lengthening and the greater convexity of the cornea as causes of myopia. The deep anterior chamber of myopic persons misled observers of that period into believing that the cornea exhibited an abnormal convexity. Morgagni,[4] in 1761, also gave an anatomic demonstration of the greater length of the myopic eye. Guérin,[2] in 1769, first mentioned the ectasia of the posterior pole. Scarpa,[5] in 1801, introduced the term staphyloma posticum but did not associate it with myopia. In the dissection of many myopic eyes, von Arlt,[3] in 1854, found that elongation at the expense of the posterior wall was the usual occurrence, and he can be credited with proving the relationship between increased length of the eye and near sightedness. The increased length was still confused with other factors, however; and a thicker lens, a more convex cornea and increased coefficients of refraction of the various media were equally considered as factors in the production of myopia. Even von Graefe,[6] prior to the introduction of the ophthalmoscope, expressed the belief that the cause of myopia was to be discovered in the vitreous humor. Finally, Donders [7] demonstrated, by ophthalmometric measurements, that the

From the Institute of Ophthalmology, Columbia-Presbyterian Medical Center.

1. Kepler, cited by Donders,[7] pp. 444-446.
2. Cited by Parsons,[118] p. 908.
3. Cited by Donders,[7] p. 447.
4. Morgagni, J. B.: The Seats and Causes of Diseases Investigated by Anatomy, translated from the Latin by B. Alexander, London, Miller and Cadell, 1769, p. 299.
5. Scarpa, cited by Donders,[7] pp. 371 and 447.
6. von Graefe, A., cited by Donders,[7] pp. 447-448.
7. Donders, F. C.: On the Anomalies of Accommodation and Refraction of the Eye, translated into English by W. D. Moore, London, New Sydenham Society, 1864, pp. 332-448.

myopic cornea is as flat as, or even flatter than, the normal cornea and proved that the axial form of myopia is dependent on a lengthening of the optic axis.

The ophthalmoscope proved a remarkable stimulus to the development of ophthalmology in general, and myopia shared in the general enlightenment that ensued. A host of papers were written on the subject of myopia in the years immediately following von Helmholtz' introduction of the instrument. The description of the subject, presented by Donders [7] in his masterpiece in 1864, constitutes an excellent résumé of the opinions generally held at that time.

Donders wrote:[7]

> The distribution of Myopia, chiefly among the cultivated ranks, points directly to its principal cause: Tension of the eyes for near objects. Respecting this fact there can be no doubt. But the explanation of it is not so evident. . . . How then is this prolongation [of the visual axis] to be explained? Three factors may here come under observation: first, pressure of the muscles on the eyeball in strong convergence of the visual axes; second, increased pressure of the fluids, resulting from accumulation of blood in the eyes in the stooping position; third, congestive processes in the fundus oculi, which, leading to softening, even in the normal, but still more under the increased pressure of the fluids of the eye, give rise to extension of the membranes. That in increased pressure the extension occurs principally at the posterior pole, is explained by want of support from the muscles of the eye at that part.
>
> Now in connexion with the causes mentioned, the injurious effects of fine work is, by imperfect illumination, still more increased: for thus it is rendered necessary that the work be brought closer to the eyes, and that consequently the convergence be stronger, and the tendency to stooping position of the head, particularly in reading and writing, is also increased. To this it is to be ascribed, that in schools, especially in boarding schools, where, by bad light the pupils read bad print in the evening, or write with pale ink, the foundation of Myopia is laid, which in fact, is usually developed during these years. . . . The same causes, which give rise to Myopia, are still more favorable to its further development.
>
> Having once occurred, the Myopia is often transmitted as predisposition to posterity, and under fresh exciting causes is developed to its higher degrees. Thus the hereditary principle accumulates in the posterity the effect of the causes repeated in every generation. In some families the Myopia therefore has attained a high degree, and the danger is greater, because, according to experience, the hereditary tendency manifests itself the more certainly, the more the myopia has already been transmitted through a number of generations, and has assumed a typical character.

A legion of papers have been written about the pathogenesis and the pathology of myopia since the publication of Donder's book, but most of them have reiterated one or another of the causes he propounded. These are five in number: (1) excessive convergence, (2) excessive accommodation, (3) heredity, (4) increased pressure of the fluids with bending the head, and (5) congestion of the coats of the eye, leading to softening and ectasia. Other factors which have

been advocated are: (1) too short an optic nerve (2) congenital deficiency of the sclera, (3) disorder of growth, (4) imbalance of the extraocular muscles, (5) psychic and intellectual relation, (6) endocrine dysfunction, (7) avitaminoses, (8) constitutional diseases, (9) biologic variation and (10) sclerochoroiditis.

ETIOLOGIC FACTORS IN MYOPIA

Excessive Convergence.—The first theory to receive serious consideration as the cause of myopia was that of excessive convergence. Von Arlt [8] expressed the opinion that the pressure of the extraocular muscles in convergence impeded the outflow of blood from the eye through the vortex veins, resulting in congestion and increased intraocular tension. He regarded the external rectus and the inferior oblique muscles as the main offenders. Von Helmholtz [9] expressed a similar opinion, and he wrote in 1856 that looking at near objects and converging too much causes stretching, pulling and distention of the eye from increased blood pressure and muscular strain. Von Graefe,[10] concurring, stated the opinion that the lateral and medial rectus muscles were responsible. This led to a series of prophylactic and therapeutic tenotomies of the medial rectus muscles. Gerloff [11] even recommended tenotomy of both lateral and medial rectus muscles. Stilling [12] incriminated the superior oblique muscle and made an extensive survey of the problem. He taught that myopia varied with the position of the trochlea; if it was low, the globe was considerably compressed. He tried to prove that the orbit, and so the pulley, is low and broad in myopic persons, and he divided people into types according to the bony configuration of their orbits. Baer,[13] Schmidt-Rimpler,[13] Seggel,[13] Herrnheiser [13] and Weiss [13] duplicated his studies but failed to confirm his results. Müller,[14] believing the inferior oblique to be the cause, tenotomized this muscle in 21 patients and reported decrease in the myopia, retrogression of the chorioretinal changes and improvement in the visual acuity.

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8. von Arlt, C. F.: Ueber die Ursachen und die Entstehung der Kurzsichtigkeit, Vienna, Wilhelm Braumüller, 1876.

9. von Helmholtz, H.: Handbuch der physiologischen Optik, Ithaca, N. Y., translated and published by Optical Society of America, 1924, vol. 1, p. 141.

10. von Graefe, A.: Beiträge zur Physiologie und Pathologie der schiefen Augenmuskeln, Arch. f. Ophth. **1**:1-167, 1854.

11. Cited by Parsons,[118] p. 924.

12. Stilling, J.: Ueber das Wachsthum der Orbita und dessen Beziehungen zur Refraction, Arch. f. Augenh. **22**:47-60, 1891.

13. Cited by Parsons,[118] p. 925.

14. Müller, L.: Ueber Pathogenese und Behandlung der Kurzsichtigkeit und ihre Folgen, Wien. klin. Wchnschr. **39**: 321-325, 1926.

Other investigators have attributed myopia to excessive convergence, omitting the factor of increased intraocular pressure. Foerster [15] taught that myopia and its tendency to increase were directly due to excessive convergence of the visual axes. He asserted that it could be prevented by use of full correction or with abducting prisms, and he published notes on 51 cases in which cure was effected with this method. In this country, Edward Jackson [16] was a staunch supporter of Foerster's theory and published a number of papers on the subject. Jackson [16c] wrote:

> Myopia's pathology begins, continues, and is most extensive at the temporal side of the optic nerve entrance. The myopic crescents and atrophies have this location. The main obstacle to free movement of the eyeball is the rigid optic nerve, firmly attached to the sclera. In convergence this resistance is constantly added to that of opposing muscles and other tissues. On the nasal side, the tissues are jammed together by the turning in of the eyeball. On the temporal, they are put on the stretch, and the choroid is pressed between the sclera and the contents of the globe. This results in the pathology seen in the myopic fundus. They are the constant and essential lesion of myopia.

He also advocated control of myopia by full correction with concave lenses or with abducting prisms. Harlan,[17] Lancaster,[18] Dvorak,[19] Luedde [20] and many others have been proponents of this theory.

Excessive Accommodation.—The earliest proponents of accommodation as the cause of myopia stated the belief that the intraocular tension was raised during accommodation. Kepler,[1] Ware,[21] von Arlt,[3] Donders,[7] Erismann,[22] Dobrowolsky,[11] Mauthner,[11] Mackenzie [23] and Junge [24] expressed this belief. Hess and Heine [25] disproved this

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15. Foerster, R.: On the Influence of Concave Glasses and Convergence of the Ocular Axes in the Increase of Myopia, translated by J. A. Spaulding, Arch. Ophth. **15:** 399-435, 1886.

16. Jackson, E.: (*a*) The Full Correction of Ametropia, Tr. Sect. Ophth., A. M. A., 1891, p. 133; (*b*) The Full Correction of Myopia, Tr. Am. Ophth. Soc. **6:** 359-373, 1892; (*c*) The Control of Myopia, Am. J. Ophth. **14:** 719-725, 1931; (*d*) Control of Myopia, J. A. M. A. **105:**1412-1416 (Nov. 2) 1935.

17. Harlan, G. C.: Constant Correction of High Myopia, Tr. Am. Ophth. Soc. **6:** 374-387, 1892.

18. Lancaster, W. B., in discussion on Jackson.[16d]

19. Dvorak, J. E.: Present Status of the Management of Myopia, J. Iowa M. Soc. **26:** 25-31, 1936.

20. Luedde, W. H.: Monocular Cycloplegia for the Control of Myopia, Am. J. Ophth. **15:** 603-610, 1932.

21. Ware, J.: Observations Relative to the Near and Distant Sight of Different Persons, Phil. Tr. Roy. Soc., London, 1813, p. 31.

22. Erismann, F.: Ein Beitrag zur Entwicklungs-Geschichte der Myopie, Arch. f. Ophth. **17:** 1-56, 1871.

23. Mackenzie, W.: Practical Treatise on the Diseases of the Eye, Boston, Carter, Hender & Co., 1833, pp. 593-604.

tenet by demonstrating that even maximal contraction of the ciliary muscle does not result in increased intraocular tension. Duke-Elder [26] cited considerable evidence produced by a number of investigators to prove that accommodation and convergence have no influence on the tension clinically. Moreover, accommodation is thought to assist in lowering the intraocular pressure by constricting the anterior ciliary arteries, by opening up the choroidal veins and by opening the angle of the anterior chamber. Ochi [27] produced artificial compression of the globes in experimental animals, by increasing the compression of the extraocular muscles, in an attempt to produce axial lengthening but was unsuccessful. Further, some observers have reported a connection between myopia and hypotension (Duke-Elder,[28] Lagrange,[29] Urio [29]).

Newman [30] attributed the development of myopia to the effects of excessive accommodation on the choroid. He stated that accommodation pulls on the elastic membrane of the choroid, tensing it and decreasing its nutrition, so that it may give way and staphyloma of the eye result. Hensen and Volkers [24] showed that needles passed into the choroid back of the equator move little on accommodation, and around the posterior pole not at all.

The most popular of all the theories of myopia, even to the present day, has been Cohn's idea of school myopia. Cohn postulated that myopia was caused by near work in school and gave as proof the fact that myopia increases in frequency and in degree during the school years. He found that 1.4 per cent of children had myopia in the lower grades of school, a proportion that steadily rose to about 60 per cent among the university students. Cohn's view had previously been expressed by Donders.[7] It was the discovery of the incidence of myopia in schools that initiated the revolution in school hygiene. It led to the insistence on clean schools, well lighted rooms, graded desks, good print, limitation of fine work, examination for ocular defects and the provision of treatment for these defects. If one accepts Cohn's figures at their face value, there would seem to be no need of further occurrence of myopia; the disease should be eradicated. Hogarth [31]

24. Cited by Parsons,[118] p. 923.

25. Hess, C., and Heine, L.: Arbeiten aus dem Gebiete der Akkommodationslehre, Arch. f. Ophth. **46**:243-276, 1898.

26. Duke-Elder, S.: Text-Book of Ophthalmology, St. Louis, C. V. Mosby Company, 1938, vol. 1, pp. 518 and 522-523.

27. Ochi, S.: Relation of the Ocular Muscles and Sclera in the Etiology of Myopia, Am. J. Ophth. **2**: 675-678, 1919.

28. Duke-Elder,[26] 1945, vol. 3, p. 3418.

29. Cited by Duke-Elder.[28]

30. Newman, F. A.: Acquired Axial Myopia, Am. J. Ophth. **12**: 714-719, 1929.

31. Hogarth, M., cited by Harman,[46] p. 11.

reported that in Sweden improved hygiene, good school lighting, abolition of the old Gothic type and the increase of outdoor sports were followed by a striking reduction in the incidence of myopia.

Duane [32] was one of the first to suggest that there is no such thing as school myopia; he was doubtful whether near work could cause near sightedness. Lawson,[33] who has done a good deal of work on the vision of school children, has changed his mind. In 1898 he [33a] expressed belief in the Cohn school of thought. In 1919 he [33b] refuted Cohn's hypothesis and asserted that the incidence of myopia is the same in children who do not go to school as in those who do. In an editorial in the *British Medical Journal,* he said: "We now believe that myopia is neither originated nor increased by near work." [33b] Thompson [34] reported some contradictory statistics in 1919; in a survey of myopia in different parts of England, he found a higher percentage of myopic persons in rural areas (24.2 per cent) than in urban areas (17.6 per cent). Gullstrand [35] denied that accommodation, convergence or near work is the cause. Parsons [36] stated that near work has no harmful effect on low and medium myopia. On the other hand, Duke-Elder [37] found that there was a larger number of cases of myopia than normal among compositors and that in a high percentage of these cases the myopia was progressive. He concluded that near work has a deleterious effect on vision and encourages the development of near sightedness. Rasmussen,[38] in a survey of 120,000 refractions in China, found 70 per cent of the people myopic, a figure comparable to that generally accepted in China for many centuries. He ascribes this high incidence to excessive accommodation and convergence in study, poor illumination, difficult Chinese characters and bending over low desks to read. Strangely enough, in the same article he observed that 98 per cent of the population were illiterate.

32. Duane, A.: Some Considerations on the Hygienic and Prophylactic Treatment of Myopia, New York M. J. **75**:983-986, 1902.

33. Lawson, A.: (*a*) Abstract of a Report on the Vision of Children Attending London Elementary Schools, Brit. M. J. **1**:1614-1617, 1898; (*b*) School Myopia, ibid. **2**:420, 1919.

34. Thompson, E.: Some Statistics of Myopia in School Children, with Remarks Thereon, Brit. J. Ophth. **3**:303-310, 1919.

35. Gullstrand, A.: Helmholtz Treatise of Physiologic Optics, Ithaca, N. Y., Optical Society of America, 1924, vol. 1, pp. 379-381.

36. Parsons, J.: Developmental Myopia and the Treatment of Myopes, Lancet **2**:796-797, 1933.

37. Duke-Elder, W. S.: An Investigation into the Effect upon the Eyes of Occupations Involving Close Work, Brit. J. Ophth. **14**:609-620, 1930.

38. Rasmussen, O. D.: Incidence of Myopia in China: Data and Theses from Periodical Investigations Covering Thirty Years Residence, and Association with Refracting and Hospital Centers, in a Score of the Larger Cities, Brit. J. Ophth. **20**:350-360, 1936.

Heredity.—Many ophthalmologists have expressed agreement with Donders that there is a hereditary factor in the causation of myopia. A few have affirmed that myopia is solely a hereditary disease; more have said that there is a hereditary predisposition. Duke-Elder [39] stated that high myopia is definitely hereditary. Gifford [40] wrote:

> The most important cause of axial myopia is hereditary, in the sense of an hereditary tendency for the eyes to reach abnormal proportions in the course of growth.

Sathaye [41] also claimed that heredity plays an important part in myopia, probably following the mendelian pattern. As a result of statistical studies of myopia in the east end of London among Jewish and non-Jewish children, Sourasky,[42] too, expressed favor toward the theory of a hereditary influence and suggested that a sex-determined factor controlled the growth of the eye. Wood [43] based his theory of a deficient ciliary muscle on the transmission of a hereditary defect. In a review of the subject, Prangen [44] wrote:

> In trying to arrive at the most plausible cause of myopia, heritage seems to be the one outstanding factor. Myopia is undoubtedly an inherited characteristic, which in all probability, follows Mendel's law.

Doyne [45] stated the opinion that the hereditary influence was questionable. Thompson [45a] was not able to establish any definite hereditary pattern. He found many cases of high myopia in families without any taint of near sightedness and, vice versa, many normal children of parents with a high degree of myopia. In an extensive study of 300 cases of high myopia, Harman [46] found a definite family history of myopia in 50 cases (17 per cent). In a later series, of 547 cases, he found such a history in 32.8 per cent. Banerjee,[47] studying the vision of students in India, collected 700 cases of myopia, in 40 per cent of which there was evidence of a hereditary influence.

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39. Duke-Elder, W. S.: Practice of Refraction, Philadelphia, The Blakiston Company, 1945, pp. 77-92.

40. Gifford, S. R.: Textbook of Ophthalmology, Philadelphia, W. B. Saunders Company, 1945, pp. 52-58.

41. Sathaye, V. D.: Myopia, Indian J. Ophth. **2**:132-138, 1941.

42. Sourasky, A.: Race, Sex, and Environment in the Development of Myopia: Preliminary Communication, Brit. J. Ophth. **12**:197-212, 1928.

43. Wood, A.: A Suggestion as to the Cause of Myopia, Ophthalmoscope **14**:302-306, 1916.

44. Prangen, A., deH.: The Myopia Problem, Arch. Ophth. **22**:1083-1096 (Dec.) 1939.

45. Doyne, P. G.: The Myopic Child, Clin. J. **52**:157, 1923.

46. Harman, N. B.: An Analysis of Three Hundred Cases of High Myopia in Children, with a Scheme for Grading Fundus Changes in Myopia, Tr. Ophth. Soc. U. Kingdom **33**:202-220, 1913.

47. Banerjee, J.: State of Vision of Indian Students in Calcutta, Calcutta M. J. **33**:53-63 and 285-295, 1938.

Increased Pressure of the Intraocular Fluids on Bending the Head Forward.—Levinsohn [48] was the most emphatic proponent of this theory. He postulated that when the head is bent forward the eye is somewhat proptosed, owing to the action of gravity and increased congestion in the orbit. This forward displacement of the globe results in a pull on the optic nerve because all the mesodermal attachments of the eye are distensible and the nerve is not. He claimed to have proved this theory experimentally in dogs, cats, rabbits and monkeys, by suspending the animals in an inverted position for several months. Essed and Soerwarno [49] confirmed his observations. Marchesani [50] repeated Levinsohn's experiments on apes, using a control group, and found myopia as often in the controls as in the subjects. Comberg,[51] in measurements on corpses, observed that there is 5 mm. of possible lengthening of the optic nerve, and he stated that the globe can never "hang" on the optic nerve in the sense that Levinsohn meant. Bucklers[52]; Scheerer and Seitzer,[53] and Jablonski [54] all repeated Levinsohn's experiments but failed to confirm his results.

Edridge-Green [55] favored a somewhat similar principle. He stated the belief that bending, straining and lifting increased the intraocular pressure by obstructing the flow of lymph out of the eye, and so causing distention of the sclera. Galloway [56] agreed with Edridge-Green and explained the incidence of myopia in laborers, farm servants and other heavy workers in this way. Lipschutz,[57] too, explained myopia as the result of increased intraocular pressure, due to bending the head forward.

48. Levinsohn, G.: Notes on the Genesis of Myopia, Arch. Ophth. **54**:434-439 (Sept.) 1925; Reply to Criticisms of My Theory on the Genesis of Myopia, ibid. **15**:84-85 (Jan.) 1936.

49. Essed, W. F., and Soerwarno, M.: Ueber Experimentalmyopie bei Affen, Klin. Monatsbl. f. Augenh. **80**:56-62, 1928.

50. Marchesani, O.: Untersuchungen über die Myopiegenese (Die experimentelle Affenmyopie), Arch. f. Augenh. **104**:177-191, 1931.

51. Comberg, W.: Anatomic and Experimental Examinations of the Mechanical Factors in the Origin of Myopia, Arch. Ophth. **1**:286 (Feb.) 1929.

52. Bucklers, M.: Anatomische Untersuchungen über die Bezeichungen zwischen der senilen und der myopischen Circumpapillaren, Arch. f. Ophth. **121**: 243-283, 1928.

53. Scheerer, R., and Seitzer, A.: Ueber das Auftreten von sogenannten myopischen Veränderungen am Augenhintergrund bei den verschiedenen Brechungszuständen des Auges, Klin. Monatsbl. f. Augenh. **82**:511-515, 1929.

54. Jablonski, W.: Ueber den augenblicklichen Stand der Erforschung der Kurzichtigkeit, Zentralbl. f. d. ges. Ophth. **28**:129, 1932.

55. Edridge-Green, F. W.: The Cause and Prevention of Myopia, Lancet **1**:136-137, 1918; Cause and Prevention of Myopia, ibid. **1**:469-471, 1921.

56. Galloway, A. R.: The Education of Myopes, Brit. M. J. **2**:46, 1923.

57. Lipschutz, H.: Myopia and Near Work, Brit. J. Ophth. **19**:611-612, 1935.

Congestion of the Coats, Leading to Softening.—Three men, in addition to Donders,[7] suggested this factor as the etiologic process. Norris [58] asserted that abuse of the eyes leads to congestion, infiltration of the tunics of the eye with serum and softening, so that the protective coats may give way under normal intraocular pressure. Turner [59] theorized that toxins from a diseased condition of the nose and throat cause chronic waterlogging of the choroid and sclera, as well as an increase in the bulk of the vitreous, and push the sclera backward without any change in the tension. Lindner [60] expressed the belief that in near work there is a transudation of the blood elements into the tissues about the choroidal blood vessels. This transudate causes a lowering of the resistance of the sclera by some enzymatic action. He stated the belief that the suprachoroidea serves as a protective layer for the sclera and explained the localization of myopic lesions at the posterior pole on the basis of the thinning of the suprachoroidea posteriorly. A contributing factor, Lindner suggested, is the increased transudation about the posterior pole because of the increased thickness of the choriocapillaris around the fovea.

Too Short an Optic Nerve.—A tenet that attained some popularity about fifty years ago was the belief that the optic nerve was too short in myopic persons and so pulled on the posterior pole of the globe. This idea was first suggested by Hasner,[18] and the theory was fostered particularly by Weiss.[61] Emmert,[62] Paulsen [63] and Thorner [64] also advocated it. Stilling [13] examined and measured a series of 200 optic nerves post mortem and found the optic nerve short in very few eyes, and those few were not myopic. Schnabel,[65] Hess,[66] Comberg,[51] Hanssen [67]

58. Norris, W. F.: Some Remarks on Asthenopia and the Changes in Refraction in Adolescent and Adult Eyes, Tr. Am. Ophth. Soc. **4**:369-384, 1886.

59. Turner, H. H.: Discussion of the Elements Underlying Progressive Axial Myopia, Pennsylvania M. J. **34**:173-178, 1930; The Etiology and Control of Progressive Axial Myopia, ibid. **47**:793-801, 1944.

60. Lindner, K.: Ueber den Einfluss von Umvelt und Vererbung auf die Entstehung der Schulmyopie, Arch. f. Ophth. **146**:336-376, 1946.

61. Weiss, L.: Beiträge zur Entwicklung der Myopie, Arch. f. Ophth. **22**: (pt. 3):1-124, 1876

62. Emmert, E.: On the Causes of Myopia, Ophthalmoscope **2**:374-375, 1904.

63. Paulsen, O.: Ueber die Entstehung des Staphyloma posticum chorioideae, Arch. f. Ophth. **28**:225-244, 1882.

64. Thorner, cited by Luedde.[20]

65. Schnabel, J.: Zur Lehre von dem Ursachen der Kurzsichtigkeit, Arch. f. Ophth. **20**(pt. 2):1-70, 1874.

66. Hess, C.: Ueber den Einfluss, den der Bruchungsindex des Kammerwassers auf die Gesammt-Refraction des Auges hat, Klin. Monatsbl. f. Augenh. **36**:274-280, 1898.

67. Hanssen, R.: Zur Genese der Myopie, Klin. Monatsbl. f. Augenh. **67**: 171-172, 1921.

and von Hasses [68] could not agree with Hasner and Weiss. In fact, von Hasses suggested that too long an optic nerve might be the cause! He observed a very long and sinuous optic nerve, resembling a spiral behind the globe, in many myopic persons, and proposed that backward movement of the globe on blinking the lids may weaken the posterior pole by causing it to hammer against this springlike optic nerve.

Congenital Deficiency of the Sclera.—Mauthner [69] initiated the proposal that the staphyloma of myopia is due to congenital weakness of the sclera. This concept was then taken up by Schnabel and Herrnheiser,[70] who endeavored to prove it anatomically. They showed that when an emmetropic eye is placed on a flat surface with the cornea down, the eye keeps its shape. However, when a myopic eye is placed in a similar position, some part of the posterior pole will dimple inward. Luedde,[20] Henderson,[71] Dean,[72] Couadau [73] and Incze [74] endorsed this explanation of the pathologic basis of myopia. Incze stated that there is an asthenic tendency in myopia, in that the scleral weakness is part of a general weakness of all mesoderm-derived tissues throughout the body. According to Incze, distention of the eye results from daily fluctating hypertonia on the weak sclera.

Disorder of Growth.—That myopia is an overgrowth of the eyeball was proposed by Vogt [75] in 1923. He stated that the retina is the determinative factor in the size of the eye and that myopia results when the retina has too much inherent growth potential. He compared the growth of the eye around the retina with the growth of the skull around the brain. Stocker [76] endorsed Vogt's theory and attempted to explain the pathologic changes at the posterior pole on that basis.

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68. von Hasses, cited by Poos, F.: The Myopic Problem, Arch. Ophth. **2**:66-75 (July) 1929.

69. Cited by Parsons,[118] p. 926.

70. Schnabel, J., and Herrnheiser, I.: Ueber Staphyloma posticum, Konus und Myopie, Ztschr. f. Heilk. **16**:1-48, 1895; abstracted, Jahrb. u. Ophth., 1896, pp. 436-437.

71. Henderson, T.: The Constitutional Factor in Myopia, Tr. Ophth. Soc. U. Kingdom **54**:451-459, 1934.

72. Dean, F. W.: Axial Myopia: Phases of Etiology and Treatment, J. Iowa M. Soc. **21**:216-220, 1931.

73. Couadau, A.: La myopie monolatérale: Travail de la clinique opthalmologique du Prof. Frankel, Paris, J. B. Baillière, 1929, p. 119.

74. Incze, A.: Ueber die Myopie als eine konstitutionelle Veränderung, Ztschr. f. Augenh. **67**:20-41, 1929.

75. Vogt, A.: Ueber Vererbung von Augenleiden, Schweiz. med. Wchnschr. **53**:188-193, 1923.

76. Stocker, F. W.: Pathologic Anatomy of the Myopic Eye with Regard to Newer Theories of Etiology and Pathogenesis of Myopia, Arch. Ophth. **30**: 476-488 (Oct.) 1943.

According to Stocker, when the retina grows too much on the nasal aspect of the disk, supertraction ensues, and when it does not grow quite enough on the temporal side, the myopic crescent results.

Holm [77] theorized that the tonus of the ciliary muscle is a regulating influence, directing the growth of the eye. Therefore, the increased innervation of the ciliary body required during long periods of reading acts as a growth-promoting factor, and school myopia is the common result. Progress beyond 3 D. of myopia is explained by superinnervation of the ciliary muscle; the myopic student brings his book closer than his near point because convergence to that point stimulates his accommodation; and the closer he moves it, the more accommodation he needs.

Wood [43] held that myopia is due to lack of development of the ciliary muscle, particularly of Müller's fibers. This defect, usually described as a result of myopia, is present at birth, according to Wood; he stated, therefore, that it cannot be a phenomenon of disuse. Keith [78] stated the belief that it is a growth disorder, occasioned by the conditions which civilization has imposed on man. He compared myopia with acromegaly, with the idea that some comparable fault in the mechanism that regulates the growth of the sclera must be at the root of myopia. Posey [79] suggested that the shape of the skull determines the conformation of the orbit and, in turn, the form of the globe. Myopia, according to Posey, is common in races like the Teutons, whose skulls are long anteroposteriorly, and is rare in the Negro and the Indian, of whom the reverse is true. Vandergrift [80] concurred with Posey in this belief and stated that myopia is the price paid by man for his high mentality. Walker [81] and Duke-Elder [82] also expressed the opinion that axial myopia is an aberrant growth process.

Imbalance of the Extraocular Muscles.—Marlow [83] observed that imbalance of the extraocular muscles is present in a high percentage of myopic eyes and suggested that this is an important, probably the most important, active factor in the onset and progress of this condition.

77. Holm, E.: The Pathogenesis of Reading Myopia, Acta ophth. **3**:234-244, 1926.

78. Keith, A.: Myopia, a Disorder of Growth, Lancet **1**:32-33, 1925; On the Nature of Man's Structural Imperfections, ibid. **2**:1047-1051, 1925.

79. Posey, W. C.: Hygiene of the Eye, Philadelphia, J. B. Lippincott Company, 1918, p. 32.

80. Vandergrift, G. W.: The Development of the Accommodative Apparatus in Relation to Myopia and Presbyopia, New York State J. Med. **24**:385-389, 1924.

81. Walker, J. P.: Myopia and Pseudo-Myopia, Brit. J. Ophth. **30**:735-742, 1946.

82. Duke-Elder,[39] p. 79.

83. Marlow, F. W.: Muscle Imbalance in Myopia, Arch. Ophth. **13**:584-597 (April) 1935.

He advised that an occlusion test for latent muscular imbalance be carried out in all cases as early as possible. Pascal [84] agreed that exophoria may produce myopia by compression of the extraocular muscles, and also by excessive stimulation of accommodation due to overstimulation of convergence. In a statistical study of functional muscle tests in axial myopia, Snell,[85] however, found nearly twice as much esophoria as exophoria. In his series of 1,078 cases of myopia, esophoria was exhibited in 55.3 per cent and exophoria in only 30.4 per cent. He concluded that convergence insufficiency is not a specific characteristic of myopia. In a survey of 790 cases of myopia, Thompson [34] found divergent squint in 29 cases (3.67 per cent) and convergent deviations in 45 cases (5.69 per cent).

Psychic and Intellectual Relations.—Straub [86] maintained that the psyche is responsible for the state of refraction. Only those who wish to see well at a distance are emmetropic, and those who are overly attracted to near objects become myopic. Drualt-Tonfesco [87] regarded a person's refraction as part of his personality and mental makeup. He claimed that myopic and hypermetropic persons do not reason, write, work or draw in the same way as emmetropic persons and that one's refraction can be determined by the style of one's writing or painting. Mills,[88] calling attention to the high incidence of myopia among the world's great painters, and also in a private series of artists in his own practice, asserted that there is a connection between the education and myopia. He found a close relation between near sightedness and high mental speed, intense aspiration and the severe competition of civilization. Heinonen [89] also found myopia bound up with the intellectual endowment.

Endocrine Dysfunction.—Among the more modern explanations of myopia, that of an endocrine disturbance has been approved by several authors. Wiener,[90] in the United States, has been the strongest advocate

84. Pascal, J. I.: Myopia and Exophoria, Arch. Ophth. **14**:624-626 (Oct.) 1935.

85. Snell, A.: A Statistical Study of Functional Muscle Tests in Axial Myopia, Tr. Sect. Ophth., A. M. A., 1936, pp. 49-64.

86. Straub, M.: La part de l'hérédité et de l'étude dans le développement de la myopie, Arch. d'opht. **36**:68-70, 1919.

87. Drualt-Tonfesco, S.: Notes sur la myopie, Ann. d'ocul. **159**:709-729 and 865-888, 1922.

88. Mills, L.: Peripheral Vision in Art, Arch. Ophth. **16**:208-219 (Aug.) 1936.

89. Heinonen, O.: Entsteht die Schul- und Berufsmyopie infolge der Näharbeit, oder gibt es andere Faktoren, welche siche an erster Stelle hierbei geltend machen? Acta ophth. **6**:238-250, 1928.

90. Wiener, M.: Epinephrine in Progressive Myopia, J. A. M. A. **89**:594-596 (Aug. 20) 1927; The Use of Epinephrine in Progressive Myopia: Further Report, Ann. J. Ophth. **14**:520-522, 1931; Myopia: Cause, Progress and Treatment, South. M. J. **24**:529-534, 1931.

of this hypothesis and has actively sponsored instillation of epinephrine in the eyes as a mode of therapy. In 1931 he reported results of this type of treatment in a series of 99 cases of actively progressive myopia. In 79 cases there was no further increase in refractive error after one year, while in 20 the myopia was still progressive. Wiener described the case of an intelligent woman who, when she notices her distant vision failing, instills more epinephrine drops and can see better in a very short time. Nicolato [91] also expressed the opinion that endocrine disturbance, especially a thymic dysfunction, is the hereditary cause of all myopia. Malone [92] favored an endocrine upset as the cause but did not state which gland was involved; he also reported beneficial results from epinephrine. Haseltine [93] expressed the opinion that progressive myopia and glaucoma are both the result of an endocrine disturbance, and reported 5 cases in which progressive myopia was definitely improved by administration of a preparation of the adrenal gland. Costello [94] described a syndrome of hypothyroidism and myopia. Bothman [95] also suggested that hypothyroidism may be a cause of myopia. He found a low basal metabolic rate in a series of cases of progressive myopia and reported that in some cases the condition ceased to progress after thyroid was given. Law [96] reported favorable effects from calcium and parathyroid therapy.

Avitaminoses.—Lack of essential vitamins as a causative factor in myopia has had its adherents. Knapp [97] proposed that a disturbance in vitamin D–calcium–phosphorus metabolism was the cause of myopia. He suggested that lack of calcium may weaken the fibrous tunics of the eye. He reported beneficial optical results from vitamin D therapy, with some resolution of the pathologic process in the fundus and shrinkage of the globe. Laval [98] was not able to see any improvement in clinical

91. Nicolato, A.: Della genesi della myopia: Note preventiva, Arch. di ottal. **36**:453-468, 1929.

92. Malone, J. Y.: Progressive Myopia, Keratoconus, and Keratoglobus, Tr. Sect. Ophth., A. M. A., 1939, pp. 44-52.

93. Haseltine, S. L.: Suprarenal Gland in the Treatment of Glaucoma, Progressive Myopia, and Some Allergic Conditions, J. M. Soc. New Jersey **34**: 729-731, 1937.

94. Costello, J. P.: Obesity and Ocular Symptoms in Mentally Alert Children Due to Hypothyroidism, Endocrinology **20**:105-106, 1936.

95. Bothman, L.: The Relation of Basal Metabolic Rate to Progressive Axial Myopia: Preliminary Report, Am. J. Ophth. **14**:918-924, 1931.

96. Law, F. W.: Calcium and Parathyroid Therapy in Progressive Myopia, Tr. Ophth. Soc. U. Kingdom **54**:281-290, 1934.

97. Knapp, A. A.: Vitamin-D Complex in Progressive Myopia: Etiology, Pathology and Treatment, Am. J. Ophth. **22**:1329-1337, 1939.

98. Laval, J.: Vitamin D and Myopia, Arch. Ophth. **19**:47-53 (Jan.) 1938; The Relationship Between Myopia and Avitaminosis, Am. J. Ophth. **24**:408-412, 1941.

trials with vitamin D and concluded that there is no proof that avitaminosis causes myopia. Sathaye [41] agreed with Knapp and advised calcium and vitamin D therapy.

Constitutional Diseases.—Inevitably, syphilis and tuberculosis must be mentioned in any discussion of the pathogenesis of a disease. Kassas [99] indicted syphilis, tuberculosis and measles as predisposing factors in the causation of myopia. The inciting factor, in his opinion, is solely a poor hygienic condition. Sonder [100] stated the belief that the diseases of childhood are the causes of near sightedness. He based his opinion on his series of 100 cases of progressive myopia, in which every child had had one or more of the diseases of childhood. Myopia is a stigma of constitutional weakness, according to Henderson [71]; he suggested the phrase "scleral rickets" and said that therapy should be general, directed toward improving the constitution of the patient. In Morse's opinion,[101] rickets, chronic tonsillitis, intestinal disturbances, endocrine dysfunction and toxic colitis may all produce myopia. Miller [102] concluded that myopia is largely due to a dietary deficiency in fats. Walker [103] suggested that it is due to a calcium deficiency. The devitalizing effect of a neighboring infection of the nasal sinuses is the etiologic agent, in the opinion of Lemere.[104] Rosenow,[105] in a series of experiments on animals, proved that lesions in the eye may be due to absorption of toxins in the blood from a focus of infection elsewhere in the body. Nordgren [106] expressed the opinion that in Sweden there is a striking resemblance between general hygiene and health and the frequency of myopia.

Biologic Variation.—All the authors cited up to this point have accepted the axial elongation of the eye, as expressed by Donders, as

99. Kassas, J.: Ueber Veränderung der Refraction bei 90 Zoglingen des Brester Knabengymnasiums in 5 Jahren, West. Ophth. **30**:829, 1914; abstracted, Klin. Monatsbl. f. Augenh. **17**:754, 1914.

100. Sonder: De l'influence des maladies infantiles dans l'evolution de la myopie progressive, Arch. d'opht. **37**:290-298, 1920.

101. Morse, S.: Myopia as a Disease That May Be Preventable, Am. Med. **34**:115-121, 1928.

102. Miller, H.: Is Myopia a Deficiency Disease? Am. J. Ophth. **23**:296-305, 1940.

103. Walker, J. P. S.: Progressive Myopia: A Suggestion Explaining Its Causation, and Its Treatment, Brit. J. Ophth. **16**:485-488, 1932.

104. Lemere, H. B.: Contributing Factors in the Etiology of Myopia: Preliminary Report, Tr. Am. Acad. Ophth., 1923, pp. 132-145.

105. Rosenow, E. C.: Focal Infection and Elective Localization in the Pathogenesis of Diseases of the Eye, Am. J. Otol., Rhin. & Laryng. **36**:883-895, 1927.

106. Cited by Sorsby, A.: Modern Trends in Ophthalmology, London, Butterworth & Company, Ltd., 1940, pp. 256-267.

the pathologic basis of myopia. In 1913 Steiger [107] published his theory, which contradicted this whole conception of myopia, including not only the theories of its pathogenesis, but also the previously accepted view on the very architecture of the myopic eye. He proposed the unity of all the refractive errors. To Steiger, myopia was only a biologic variation. He plotted all refractive errors as physiologic variations on a binomial curve of frequency, constructed around a mean. He asserted that myopia is merely the antithesis of hypermetropia and that people are myopic or hypermetropic by the same chance variations as people are short or tall. Steiger proposed that the curvature of the cornea (which he found to vary) and the axial length of the eye are separate, independent, freely variable components of the total refraction, that these are hereditary components and that the chance association of these free variables will produce the whole range of refractive errors. This theory was a radical departure from the orthodox school of physiologic optics. In support of this view is the fact that an axial length which will produce emmetropia with a cornea within the normal range of refractive ability will produce ametropia, either hypermetropia or myopia, with another cornea of different refractive power, but still within normal range. Later it was demonstrated by Tron [108] that the same axial distance might be a factor in a refraction ranging from +6 to —9 D. Steiger's original hypothesis of the binomial curve breaks down on two main points: (1) the excess of emmetropic persons over the theoretic calculated number and (2) the excess of myopic persons over the estimated figure.

Tron carried on Steiger's work in great detail and contributed some important pieces to the puzzle. He estimated the range of refractive power of the cornea as between 37.0 and 48.1 D. and gave a range for the refraction of the lens as between 20.1 and 34.0 D., an important factor which Steiger overlooked. He showed, moreover, that Steiger's curve holds true for myopia if eyes with a myopia of over 6 D. or eyes showing pathologic changes in the fundus are not considered. His work also showed that axial length is a factor of great importance in cases of high myopia but is practically insignificant in cases of myopia of medium and low degrees.

Steiger's and Tron's work brought out the important conclusion that low and medium errors of refraction may well be regarded as biologic variations, and not as pathologic entities. An outstanding

107. Steiger, A.: Die Entstehung der sphärischen Refraktionen des menschlichen Auges, Berlin, S. Karger, 1913.

108. Tron, E.: Variationsstatistische Untersuchungen über Refraktion, Arch. f. Ophth. **122**:1-33, 1929; Ueber die optischen Grundlagen der Ametropie, ibid. **132**:182-223, 1934.

result of their work was the new idea of classifying myopia as biologic and pathologic forms.

Parsons,[36] in 1933, expressed views that were similar to those of Steiger and Tron. He said too much significance had been attached to the length of the myopic eye and pointed out the figures given as a normal variation for the emmetropic globe. He described myopia statistically in terms of frequency variations and said that the low degrees of myopia were as normal as short stature.

Sorsby [109] also attacked the axial theory of myopia and considered the theory of biologic variation. He pointed out that the length of the globe was significant only with high hypermetropia and with high myopia of 6 D. or more. He expressed agreement with the belief that the low and medium errors of refraction are probably physiologic variations. But while he agreed that myopia is likely to occur as a hereditary biologic variable, he still pondered the problem of use and abuse of eyes as a causative factor.

Sclerochoroiditis.—When von Graefe [110] first began to study the fundus of myopic eyes with the new ophthalmoscope, he concluded that the pathologic changes he observed in the choroid and sclera at the posterior pole were the cause of the distention of the eyes. He named the process "scleroticochoroiditis posterior." As soon as the opportunity presented, he searched microscopically for evidence of previous inflammation in the fundus, but in vain. Donders and others of that period disagreed with him, and later he discarded this view. That myopia might be due to a disease process was, however, accepted by several other ophthalmologists of the nineteenth century, among them Knies,[111] Tscherning,[112] Nordenson,[69] Ferri,[113] Smith,[114] Batten [115] and Risley.[116] Tscherning divided myopia into a benign and a malignant form; the benign he held to be congenital; the malignant (over 9 D.), to be due to insidious choroiditis. Ferri attributed the rapidly developing myopia

109. Sorsby, A.: The Control of School Myopia, Brit. M. J. **2**:730-733, 1933; The Pre-Myopic State: Its Bearing on the Incidence of Myopia, Tr. Ophth. Soc. U. Kingdom **54**:459-465, 1934.

110. von Graefe, A.: Zwei Sektionsbefunde bei Sclerotico-Choroiditis posterior und Bemerkungen über diese Krankheit, Arch. f. Ophth. **1**(pt. 1):390-401, 1854.

111. Knies, M.: Ueber Myopie und ihre Behandlung, Arch. f. Ophth. **32**: (pt. 3):15-72, 1886.

112. Tscherning, M.: Studien über die Aetiologie der Myopie, Arch. f. Ophth. **29**(pt. 1):201-272, 1883.

113. Ferri, cited by Parsons,[118] p. 927.

114. Smith, Priestley: Introduction to a Discussion on the Diagnosis, Prognosis, and Treatment of Pernicious Myopia, Ophth. Rev. **20**:331-339, 1901.

115. Batten, R. D.: Myopia the Result of Constitutional Disease, Ophth. Rev. **11**:1-24, 1892.

116. Risley, S. D., in discussion on Harlan,[17] pp. 383-384.

he found in previously emmetropic adults to slow choroiditis. Priestley Smith strongly asserted the opinion that choroiditis, especially the syphilitic type, may give rise to rapid and pernicious myopia. Risley [117] expressed the opinion that the stretching of the globe is due to the choroiditis and recorded as proof 9 cases of hypermetropia he had observed passing over into myopia. Batten asserted that the typical crescent, also found in emmetropic and hypermetropic eyes, would seem to indicate that the myopic crescent is a form of choroiditis, and not the result of myopia. In 1892 he wrote [118]:

> Why, when in addition to choroiditis and vitreous changes, there is elongation of the eye, it should be held that the dilatation is the cause of the other two, it would be hard to say. . . . I contend that it is a more warrantable assumption that myopia is caused by choroiditis, than that choroiditis is caused by myopia.

Parsons,[118] in his textbook of pathology, wrote:

> It must be remembered that there is little or no difference between the ultimate effects of an inflammatory lesion and the conditions found in myopia, and that a very slight chronic inflammation may present few objective microscopical features. It may be that the lower grades of myopia are entirely congenital in origin, whilst the higher grades of so-called progressive myopia have in addition etiologically a true inflammatory factor.

In 1916, Koster [119] ascribed advanced myopia to a primary chronic infectious chorioretinitis, aggravated by prolonged near reading. He said that full correction of such myopia is a blunder; it forces the patient to use his accommodation, and any exercise of the eye is deleterious, in the face of active choroiditis. He advised treating the choroiditis by the usual methods. De Schweinitz [120] also observed that myopia may result from choroiditis, usually after an acute illness.

Sourasky,[121] who has written several illuminating papers on myopia, discussed in 1928 the presence of typical myopic crescents in cases of low hypermetropia and in cases of low myopia. He stated the belief that if the crescent is to be regarded as really due to stretching these cases represent a rapid reduction of hypermetropia from a high degree to a low degree, or even to a low myopia, in other words, as manifes-

117. Risley, S. D.: Hypermetropic Refraction Passing While Under Observation into Myopia, Tr. Am. Ophth. Soc. **4**:520-531, 1887.

118. Parsons, J. H.: The Pathology of the Eye, New York, G. P. Putnam's Sons, 1905, vol. 3, pp. 908-931.

119. Koster, W.: School Myopia, Nederl. tijdschr. v. geneesk. **1**:2329, 1916; abstracted, J. A. M. A. **67**:475-476 (Aug. 5) 1916; Progressive Myopia and Its Treatment, Nederl. tijdschr. v. geneesk. **2**:17, 1916; abstracted, J. A. M. A. **67**: 549 (Aug. 12) 1916.

120. de Schweinitz, G.: Diseases of the Eye, Philadelphia, W. B. Saunders Company, 1916, pp. 127-137.

121. Sourasky, A.: The Growth of the Eye and the Development of Myopia: A Study in the Changes of Refraction During the School Period, Brit. J. Ophth. **12**:625-644, 1928.

tations of this process in hypermetropic eyes. According to Sourasky, then, the extensive pathologic changes sometimes found in the eyes of patients with low degrees of myopia represent stretching of a considerable degree in originally hypermetropic eyes. He mentioned the changes in the vitreous frequently associated with myopia and suggested that they point to a chronic inflammatory condition of the eye. Sourasky stated that he would like to say:

> A myopic eye elongates not because it is myopic, but because it is due to some affection. Myopia, per se, is no more the cause of the progressive elongation of the globe, than a glaucomatous cup is the cause of increasing tension.

In 1928, Dimitry [122] recalled and concurred in von Graefe's original proposition, namely, that myopia is the result of sclerochoroiditis in the posterior pole of the eye. Hallett,[123] in 1931, made the same affirmation and offered disease of the nasal sinuses as the original focus of infection. He cited Rosenow's work [105] demonstrating that ocular disease may be due to absorption of toxins from the blood stream. Finally, Cowan,[124] in 1941, emphatically called attention to the necessity of differentiating refractive myopia from the myopia associated with pathologic changes in the fundus. If a patient with choroiditis is hypermetropic, the state of the refraction is disregarded, according to Cowan, but if the patient is myopic, a diagnosis of myopia is made. A review of the pathologic changes in the fundus associated with this condition is in order.

PATHOLOGIC CHANGES ASSOCIATED WITH MYOPIA

A detailed study of the lesions in myopic eyes is not within the province of this paper. Since "sclerochoroiditis" is one of the commonest pathologic states seen by the ophthalmologist, its pathologic features should be well known to every one. It is more pertinent, then, to mention briefly the changes observed and discuss their significance.

Macroscopically, the striking feature presented by a globe with myopia is axial elongation. Wolff [125] reported a case in which the eye measured 54 mm. from cornea to posterior pole. Heine [126] demonstrated that the pathologic process causing this lengthening lies in the posterior pole of the globe. This distention of the posterior pole, called staphyloma verum by von Graefe, may be a generalized process involving the whole posterior portion of the eye, with no delineation of its borders, or, more commonly, it may be limited to a definite ectasia, usually con-

122. Dimitry, T. J.: Myopia Is Essentially a Pathological Condition, New Orleans M. & S. J. **81**:432-436, 1928.

123. Hallett, De.W.: The Prevention of Myopia, Am. J. Ophth. **14**:143-146, 1931.

124. Cowan, A.: Myopia, Tr. Am. Acad. Ophth. (1941) **46**:197-205, 1942.

125. Wolff, E.: Pathology of the Eye, Philadelphia, P. Blakiston's Sons & Co., 1935, p. 258.

126. Heine, L.: Beiträge zur Anatomie des myopischen Auges, Arch. f. Ophth. **38**:277-290, 1899.

centric with the disk. The presence of staphyloma is not constant; extensive pathologic changes in the fundus are also seen in eyes with an axial length well within normal limits.

Grossly, section of a myopic eye will also usually show an atrophic, thin ciliary muscle, due to underdevelopment or complete lack of Müller's circular fibers. As a result, the ciliary processes are less prominent and are displaced somewhat posteriorly. As another consequence, the root of the iris is situated farther back than usual, and therefore the anterior chamber appears deeper in myopic persons.

Ophthalmoscopically, the commonest finding in the fundus is the so-called myopic crescent. This crescent (of distraction) is usually temporal to the papilla but may occur anywhere around the disk. It may surround the disk, forming what is more properly called a conus. Just as the retina and choroid seem pulled away from the disk temporally, they are often found pulled over on top of the disk nasally, forming the crescent of supertraction. This is not commonly seen, for it is usually composed of all the layers of the retina and choroid, and so is indistinguishable from the surrounding fundus. Real and apparent distortion of the disk are often observed. Real distortion of the disk results from oblique insertion of the nerve, from traction of different nerve bundles as the globe distends and from crescents of supertraction. Apparent distortion occurs because the point of greatest elongation is the posterior pole, with consequent backward displacement of the temporal side of the disk. Through the ophthalmoscope, then, the nasal edge of the disk is closer to the observer than the temporal, and the papilla appears oblique.

Nearly as common as the crescent is the choroidal atrophy seen at the posterior pole. This usually begins about the disk and the fovea and, as the process becomes severer and more extensive, these two areas often fuse, forming a large centrocecal patch. This process may go on to complete degeneration of the choroid, so that the nearly bare sclera shines through. As in other forms of choroiditis, degeneration and proliferation of the pigment epithelium occur, and patches of proliferated pigment are observed in and about the areas of choroidal involvement. Choroidal hemorrhages are also frequently seen in cases of severe choroiditis. They may occur anywhere in the region of the posterior pole but, unfortunately, are most often seen in and about the fovea. Sometimes little irregular white lines are seen early in the disease. These were shown by Salzmann [127] to be minute ruptures in Bruch's membrane and, according to him, are the gates through which the choroidal process invades the retina. Less frequently, a black spot is seen at the macula. First mentioned by Foerster, it was described by

127. Salzmann, M.: Die Atrophie der Aderhaut in kurzsichtigen Auge, Arch. f. Ophth. **54**:337-410, 1902.

Fuchs in detail and is commonly called Fuchs's black spot. It was attributed to edema by Batten,[115] to hemorrhage by Collins and Mayou [128] and to piling up of pigment by Coats,[129] who published a photomicrograph demonstrating his observations.

Atrophy of the retina is secondary to the changes in the choroid. With the degeneration of the choriocapillaris, the outer layers of the retina undergo atrophy; and with the degeneration of the elastic lamina, pigment invades the retina, and eventually fusion of the whole retina and choroid into a band of connective tissue results, just as in the scarring of chorioretinitis. Because the fovea is more dependent on the choroidal circulation, degeneration is often more advanced here. Vacuolation is seen microscopically, and the process may go on to formation of cyst or hole at the macula. Peripheral cystic degeneration of the retina is thought to come on earlier and to be more extensive in myopic eyes than in the normal eye. As a result of the distention of the globe, the retinal blood vessels are stretched and assume long, straight courses.

Microscopic study of myopic globes has added little to knowledge of the cause of the lesions. The degeneration of the choroid and retina can be seen in detail, but it cannot be differentiated from the degeneration following inflammatory processes. Infiltration of small white blood cells, plasma cells and lymphocytes has been described by Parsons [118] and Salzmann.[127] The structure of the crescents can be studied; the crescent of distraction is seen to be due to uncovering of the sclera by degeneration of the choroid in varying degrees. The crescent of supertraction is seen as a fold of retina and choroid, or portions thereof. The sclera is seen to decrease steadily in thickness from the equator backward, in contrast to the condition in the normal eye. It is thinnest at the posterior pole, where it may be one-third to one-fourth the normal thickness.

Extensive pathologic changes are seen in the vitreous in this condition. Opacities are nearly always present in the active stages of the choroidal process. With enlargement of the eye, the cavity of the vitreous is necessarily enlarged, and this increase can be compensated only by transudation of fluid into the vitreous, leading to degeneration and fluidity of this structure. Detachment of the vitreous at the disk is often seen and is said to be caused by the recession of the posterior pole. The degeneration of the vitreous is accompanied with degeneration of the zonular fibers. This may result in tremulousness of the lens and iris. In such eyes the lens may be luxated spontaneously or on slight trauma. Opacities in the lens, either small, discrete ones or full-blown cataract, may also accommpany the degeneration of the vitreous.

128. Collins, E., and Mayou, M.: Pathology and Bacteriology of the Eye, Philadelphia, P. Blakiston's Sons & Co., 1925, pp. 111-121.

129. Coats, cited by Parsons,[118] p. 919.

SIGNIFICANCE OF THE PATHOLOGIC CHANGES

In assessing the part played by these pathologic processes in the causation of myopia, the main point of debate is whether the lesions in the fundus cause the lengthening of the eye or the stretching of the globe causes the lesions of the fundus. The mechanistic theories favor the latter point of view; the proponents of sclerochoroiditis, the former. Study of the pathologic picture reveals that some of the most important lesions are unexplained by stretching. The staphyloma of the posterior pole presents the first stumbling block. It is conceivable that the generalized distention of the whole posterior portion of the eye might be the result of stretching, but the commoner type of localized ectasia, in which normal sclera passes suddenly into a thin, deformed tissue, cannot be explained by stretching.

The atrophic condition of the ciliary muscle is attributed to disuse, on the basis of the observation that most myopic persons do not accommodate. The absence of this atrophy in some myopic eyes is usually explained by emmetropia or hypermetropia in the other eye, forcing the person to accommodate. It is more likely that the correct explanation lies oftener in full correction of the myopia during life, with resulting normal use of the ciliary muscle. These changes in the ciliary body are the result of the refractive condition of the myopic eye and can be seen in both physiologic and pathologic myopia. Therefore they are not part of the pathologic picture of sclerochoroiditis.

The crescent is difficult to explain. Originally every one thought it was a phenomenon of the posterior pole caused by stretching, and some said it was merely part of the staphyloma. This idea of its association with the staphyloma has been discarded. Schnabel and Herrnheiser [70] and von Szily [130] proposed that the crescent was a congenital defect, not associated with myopia. Elschnig [131] stated the belief that it was a coloboma, due to persistence of the limiting tissue between the edge of the disk and the innermost part of the pia. Parsons [132] also favored the theory of a congenital defect. Ida Mann [133] differentiated the crescent of myopia from congenital defects and stated that it was due to a slow pathologic process in the eye. If the crescent were due to distraction of the retina and choroid from the disk, it would seem that it should all be white and should not exhibit the various degrees

130. von Szily, A.: Ueber den "Conus in heterotypischer Richtung," Arch. f. Ophth. **110**:183-291, 1922.

131. Elschnig, A.: Das Colobom am Sehnerveneintritte und der Conus nach unten, Arch. f. Ophth. **51**:391-430, 1900.

132. Parsons, J. H.: Diseases of the Eye, New York, The Macmillan Company, 1942, p. 517.

133. Mann, I.: Developmental Abnormalities of Eye, London, Cambridge University Press, 1937, p. 110.

of progression, as seen in the changing colors. Further, one would not expect to see the crescent in small eyes that have not been stretched. Moreover, if the crescent were a congenital defect, one would not expect the colors to change during life. Clinical study of these crescents shows that there are all degrees of depigmentation, and thus of progression, and this observation favors Mann's theory of a pathologic process. However, the constancy of the form and shape of the crescent is against its being a purely inflammatory lesion. It seems to me that it is best explained by a combination of traction and a pathologic process: the tension on the choroid of the dilating globe is great at the posterior pole because of the insertion of the choroid into the disk, and, in the presence of weakening due to the choroidal disease, this particular part of the choroid may be prone to give way and leave the characteristic hammock defect in the choroid.

The crescent of supertraction appears to be the result of traction alone. As the globe distends at the posterior pole, there must be traction on the coats of the eye in the region of the posterior pole. The crescent of supertraction seems to be the result of this pull posteriorly. The sclera nasal to the disk is separated from the focal point of the disease process by the nerve itself, while the choroid and retina are subjected to the traction throughout their entirety.

The occurrence of the pathologic lesions in full-blown form in eyes of normal size, where stretching has been minimal or nil, is strong evidence against the mechanical school of thought. The occurrence of such extensive choroidal lesions, in itself, seems to rule against stretching, since one would not expect a tissue as elastic as the choroid to respond so drastically to tension. Further, if the choroidal lesions were due to stretching, why should they be confined to the posterior pole? It is difficult to conceive of a localized part of the choroid being stretched without affecting the rest of the choroidal tract. The late onset of the macular lesions is also evidence against stretching; they commonly come on after elongation of the eye has occurred. The infiltration of white blood cells is difficult to evaluate. Salzmann and Parsons expressed the opinion that this reaction is evidence of the irritative effect of the stretching of the globe rather than a definite sign of inflammation. This may be true; on the other hand, a similar reaction is commonly seen in sections of eyes with chronic chorioretinitis. It should be pointed out that almost the only slides of myopic eyes available for microscopic study are those of eyes removed for some other purpose, such as tumor, and often long after the active stage of the myopia. It would be of great value to study sections of myopic eyes of patients meeting death while the myopia is in the active stage. In favor of an inflammatory process are the following observations: Choroiditis is

often confined principally to the posterior pole; it usually, or perhaps always, instigates a concurrent scleritis; it is usually accompanied with opacities in the vitreous, and macular retinal changes are commonly secondary and subsequent to choroiditis. Thus, it would seem that, although some of the changes are due to traction of the distending globe, stretching per se cannot be accepted as the initial lesion in this disease.

Whether the process is an inflammatory or an atrophic one poses another problem. Fuchs [134] said the chorioretinal changes were definitely a pathologic process, but did not elaborate the idea. Troncoso [135] called the process "myopic choroidiosis" and attributed it to degeneration, following stretching of the globe, and later progressing to atrophy. The strongest evidence in favor of the inflammatory nature of the process is the clinical observation of cases in the active stage; the appearance is certainly not that of an atrophic process. It is difficult to explain the stretching of the sclera if atrophy of the choroid is accepted. The lowering of the intraocular pressure tends to support the theory of an inflammatory origin, in that this is a known accompaniment of uveal disease. It seems, therefore, that the bulk of evidence favors the inflammatory nature of the lesion and that the primary site lies in the choroid.

COMMENT

The very diversity and number of etiologic theories of the pathogenesis of myopia indicate that the problem has never been satisfactorily solved. It is difficult to account for the proposal of so many mechanical hypotheses, considering the extent of the possible pathologic changes in the myopic fundus. One can only say that too much emphasis has been placed on the element of axial elongation and that the other pathologic factors have been practically disregarded.

The theory that increased intraocular tension from excessive accommodation or convergence might cause distention of the globe has been refuted since it was first proposed. It is generally agreed that accommodation does not cause an increase in the ocular tension but, rather, is an aid in keeping the pressure within normal limits. Contraction of the extraocular muscles does cause some increase in the intraocular tension, but so little that it is not of any clinical importance. The difference between the pathology of myopia and that of glaucoma and buphthalmos seems to rule out increased tension of the eye, from any cause, as one of the etiologic factors in the production of axial elongation.

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134. Fuchs, E.: Textbook of Ophthalmology, translated by A. Duane, ed. 7, Philadelphia, J. B. Lippincott Company, 1924, pp. 193-204.

135. Troncoso, M. U.: Internal Diseases of the Eye and Atlas of Ophthalmoscopy, Philadelphia, F. A. Davis Company, 1946, pp. 463-470.

That myopia is the result of mechanical wear and tear in convergence of the eyes, as proposed by Foerster and Jackson, is unlikely. The occurrence of myopia in one eye and not in the other, and its occurrence in one-eyed people and in persons without binocular single vision are all unexplained if this theory is accepted. The more frequent association of convergence excess with hypermetropia than with myopia furnishes another objection. The arguments against school myopia also refute this hypothesis.

Donder's and Cohn's idea of school myopia is as difficult to disprove as it is to prove. One objection is the absence of definite proof that school myopia exists. The incidence of myopia during the school years may well be a mere coincidence. Statistics are available to show its similar occurrence in children who do not go to school. The increasing incidence in particular school years and the fact that myopia does not increase in colleges and postgraduate schools are difficult to associate with the theory of school myopia as the cause. Further, Brown[136] has shown that the increased incidence of myopia in the early school years conforms to the cycle of refractive changes in the normal eye. If Cohn's facts were true, the incidence of myopia should have decreased with improved school conditions. Reports of decreased incidence have issued from Sweden, Denmark, Germany and Portugal, but for every one there is a contradictory report with equally impressive statistics. This is confusing, but there has been no positive proof that the incidence of myopia has decreased. To me a strong argument against school myopia is a negative one, namely, that the incidence of myopia has not increased along with the great increase in education and literacy during the past one hundred years, with its consequent increase in the use of the eyes for near vision.

Most of the remaining theories have little in their favor; usually there are as many ophthalmologists opposed to as concurring in them. Often there seems to be a confusion between cause and result. Thus, in axial myopia there is by definition an oversize of the globe, but that is no proof that it is a disorder of growth. Likewise, with the proposal of the congenital deficiency of the sclera: The sclera becomes thin, it is true; but it does not resemble a congenital defect. The fact that myopia is commonly progressive about the time of puberty is not proof that it is an endocrine disorder.

There are two etiologic theories that are difficult to refute: One is Steiger's biologic variation, and the other, von Graefe's sclerochoroiditis. Steiger's original hypothesis has been found wanting in some respects. He neglected to include the refraction of the lens in his calculations, and

136. Brown, E. V. L.: Use-Abuse Theory of Changes in Refraction Versus Biologic Theory, Arch. Ophth. **28**:845-850 (Nov.) 1942.

he considered the refractive errors as free variables. The result was a curve that differed from the normal curve in a peak that was too high (emmetropia) and in a prolongation of the myopic limb. It was shown by Tron that exclusion of myopic subjects with lesions of the fundus changed the myopic end of the curve to the normal symmetric type. Berg [106] has since demonstrated that if a curve, calculated not on free variables, but on three correlated factors, is constructed, the result closely resembles the actual distribution of refractive errors. Thus, with the exclusion of the type associated with pathologic changes in the fundus, it is likely that Steiger was right in his description of myopia as a biologic variation, and that Donders was wrong when he said that every myopic eye is a diseased eye. The eye with the lesions of the fundus, however, is a diseased eye, and it is necessary to consider it a clinical entity in view of the large number of cases encountered with the typical characteristics. The conclusion is inevitable, then, that one must abandon the monistic schema of myopia advocated by Jackson [137] and assume that there are two main types of myopia: one, a biologic variation, and the other a pathologic disease of the eye.

CLASSIFICATION

An obstacle to the study of myopia, whether etiologic, pathologic or clinical, is the lack of a generally accepted classification. It is obvious from the literature that many ophthalmologists believe there are two types of myopia, but few classify their cases in their discussions, and most use myopia as an all-inclusive term. The result is most confusing. The factor of heredity is a representative example. Many surveys showing varying frequencies of familial histories have been reported, but without any qualification as to type of myopia. In spite of all the work done on the heredity of myopia, it is still impossible to draw any reliable conclusions.

A number of classifications have been proposed. Donders [7] classified myopia as stationary, temporarily progressive and continuously progressive forms. This division is a clinical one, based on the refractive state, is not applicable to pathologic myopia and does not differentiate between the two types. Tscherning [112] and Fuchs [134] divided near sightedness into high and low myopia, and this classification has been sponsored more recently by Rodin [138] and Stocker,[76] in this country. This division is unsatisfactory for the same reasons; it does not describe the pathologic type, and it does not differentiate the two entirely different processes. Parsons [118] set forth an anatomic classification in his

137. Jackson, E.: Norms of Refraction, Tr. Sect. Ophth., A. M. A., 1931, pp. 174-190.

138. Rodin, F. H.: Etiology of Myopia, Arch. Ophth. **9**:264-279 (Feb.) 1933.

textbook, based on the following factors: axial elongation of the globe, increased curvature of the cornea or lens, increased refractive index of the various media, forward displacement of the lens and combinations of these factors. This classification has been repeated in many textbooks, because the types cannot be diagnosed clinically; but, again, the pathologic form is not included. Prangen,[44] in 1939, suggested two main types: (1) developmental myopia, subdivided into curvature and axial forms, and (2) malignant myopia, of moderate and severe degrees. This division is not favored, because one cannot differentiate curvature myopia and axial myopia in life, and because the word "malignant" has a bad connotation. Koster [139] divided his cases of myopia into (1) a cyclitic type, with pathologic process in the fundus, and (2) an optic type, in which he considered the eye as sound as the emmetropic eye. He pointed out the importance of differentiating the two states, both for prognosis and for therapy. Sourasky [121] and Cowan [124] also vigorously sponsored this type of division but did not suggest specific terms.

The best classification is probably the simplest. Therefore it is suggested that there are two types of myopia: (1) primary, or physiologic, myopia; and (2) secondary, or pathologic, myopia. Primary myopia is, as far as is known at present, a biologic variation of refraction and a healthy one, with vision correctable to normal standards. It includes myopia of both high and low degrees, the great majority of cases being of the latter type. It is not possible to say whether the axial length or the refraction of the media is at fault, since either one can be within normal limits and still cause myopia. Secondary myopia is the result of disease of the coats of the eye. It should not be called myopia at all, but this term is proposed because of long-established custom and because the true cause is not known. The differential diagnosis of the two forms rests on the observation of pathologic changes in the fundus. The great majority of cases will be of the primary type, according to the meager statistics now available.

The results that can be obtained from the use of such a classification in a large clinic are manifold: The incidence of the two forms can be more definitely determined. The range of myopic error in the two forms can be established. The incidence of secondary myopia in previously emmetropic and hypermetropic persons can be studied. Whether the pathologic form arises more often in cases of primary myopia than in cases of emmetropia or hyperopia will be a pertinent finding. That conclusion, together with the continued use of this classification, will

139. Koster, W.: Cyclitic Myopia, Optic Myopia, and Infantile Glaucoma, Nederl. tijdschr. v. geneesk. **2**:113, 1916; abstracted, J. A. M. A. **67**:645 (Aug. 19) 1916.

demonstrate in time whether the classification is a valid one. The influence of heredity can be better studied by dividing the cases into two types. Better study and treatment of the pathologic form will evolve as a result of its separation from the errors of refraction. It is not intended that this classification is the final answer to the problem of myopia, but it is suggested that a statistical study will give much more information about myopia, when other approaches have failed.

CONCLUSIONS

1. Axial elongation of the eye beyond normal limits is not necessary to myopia.

2. Near work has never been shown to cause myopia, either in school or in adult occupations.

3. There is no proof that overcrowding, poverty, poor health, inferior or superior mentality or bad ocular hygiene can cause myopia.

4. No mechanical basis for myopia has ever been substantiated.

5. There is no evidence that congenital deficiency of the sclera, disorder of growth, imbalance of the extraocular muscles, endocrine dysfunction, or avitaminoses plays any part in the causation of myopia.

6. The relation of other diseases to pathologic myopia has not been determined. It is possible that they play a part in the production of secondary myopia.

7. The role of heredity is not definitely established, particularly with respect to secondary myopia.

8. There are statistical evidence that primary myopia is a biologic variation and clinical evidence that it is a healthy refractive state.

9. There is clinical and pathologic evidence that secondary myopia is a disease of the eye, the cause of which has not been demonstrated. It is suggested that a low grade chronic choroiditis is the primary lesion.

10. The main factor in differential diagnosis of the two types of myopia is the presence or absence of the lesions in the fundus.

11. A classification of myopia is essential for progress in statistical and clinical research on this condition. The terms primary and secondary are suggested as simple and descriptive.

12. Differential diagnosis of the two types is of great importance clinically, in therapy and in prognosis. It will prevent the waste of many useful citizens as "myopic cripples."

635 West One Hundred and Sixty-Fifth Street.

CRANIAL EPIDERMOID WITH EROSION OF THE ROOF OF THE ORBIT

Report of a Case

ISADORE GIVNER, M.D.
AND
HENRY WIGDERSON, M.D.
NEW YORK

THE FIRST primary cranial epidermoid was reported by Johannes Müller [1] in 1838. He called attention to the resemblance between cells of epidermal origin and the cells observed in these tumors. Remak, in 1854, advanced the theory of ectodermal rests to explain the origin of epithelial tumors having no attachment to the surface epithelium. Bostroem,[2] in 1897, first used the term epidermoid and pointed out the many opportunities for bits of ectoderm to be pinched off during closure of the neural tube and during subsequent stages in the development of the central nervous system, as when the transverse folds are forming. He expressed the belief that only cell rests in contact with the pia or lodged in the diploe are assured of nourishment adequate to develop into tumors. He stated that when cells destined only for the epidermis are pinched off they give rise to tumors exhibiting only epidermoid structures. When deeper layers are also included, a dermoid develops, containing hair and skin glands as well.

The term primary epidermoid is used to distinguish this tumor from collections of epithelial debris, called acquired cholesteatoma, which are observed, for example, in the ear after an infection. The term cranial distinguishes this type from the intracranial epidermoids. In the case of the epidermoids originating in the diploe of the flat bones of the skull, the increasing size of the tumor causes expansion of the overlying bone, and finally erosion, giving the typical roentgenographic picture. Bucy [3] called attention to the importance of noting the erosion of the inner table of the skull, as sometimes this erosion is only pinpoint in size and beyond

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Read at a meeting of the Section of Ophthalmology of the New York Academy of Medicine, Feb. 17, 1947. An abstract of this paper, with discussion was published in the October 1947 issue of the ARCHIVES, pages 552-554.

1. Müller, J.: Ueber den feinern Bau und die Formen der krankhaften, G. E. Schwülste, Berlin, G. Reimer, 1838.

2. Bostroem, A.: Centralbl. f. allg. Path. u. path. Anat. **8**:1, 1897.

3. Bucy, P. C.: Intradiploic Epidermoid (Cholesteatoma) of Skull, Arch. Surg. **31**:190 (Aug.) 1935.

it a larger mass of tumor may have developed between the skull and the dura. He pointed out that these minute defects may represent the persistence of the opening through which an abnormal prolongation of the papillary layer of the epidermis remained attached to the dura during the formation of the flat bones of the skull, namely, the frontal, parietal, occipital and temporal bones.

The cranial epidermoid does not infiltrate and so merely displaces brain tissue; but even though the outer layers of the tumor are friable, its growth is so inexorable that it will erode and destroy any unyielding tissue with which it may come in contact, including bone. The growth is well encapsulated and is covered with layers simulating the skin of an onion. These layers constitute the lining of the mass. The outer layer is chiefly of acellular connective tissue; under this are a middle layer, composed of stratified squamous epithelium, and an inner layer, formed of cornified epithelium.

Cushing, in a series of 2,004 cerebral tumors, found that only 0.6 per cent were epidermoids. He was one of the first to associate defects in the bones of the skull with epidermoids.[4] To date, 188 cases of cranial epidermoid have been reported. Thirty-three of the tumors were the diploic type, to which the tumor we describe here belongs. The site of election of the intracranial type is along the base of the brain and the brain stem. Over 12 per cent occurred within the ventricles. Whereas epidermoids may occur at any age, men seem to be affected more frequently than women.

REPORT OF A CASE

History and Examination.—M. N., a man aged 84, first presented himself on Oct. 22, 1946, complaining that although he had had a mass in the left frontal region all his life, for the past three years it had grown to the extent that he could not open his left eye. Examination of the right eye revealed a normal condition except that early sclerosis of the lens had reduced his vision to 20/30 with correction. In the left frontal region was a mass the size of a small apple, which fluctuated and transmitted the intracranial pulsation (fig. 1 *A*). On deep palpation around the mass, the edge of the bone could be felt, suggesting absence of the frontal bone. The lid could not be raised voluntarily, and the wall of the cyst could be felt pushing the tissues of the upper lid forward. On raising the left upper lid with the finger, one noted no elevating action of the eyeball. Intraocular examination showed nothing significant except for an immature cataract. Vision here, however, could be improved to 20/50.

Roentgenograms confirmed the complete loss of the bone in the region of the cyst and suggested absence of the roof of the orbit, the latter having been suspected on clinical grounds because of loss of ability to elevate the globe (fig. 2).

4. Cushing, H.: A Large Epidermal Cholesteatoma of Parietotemporal Region Deforming Left Hemisphere Without Cerebral Symptoms, Surg., Gynec. & Obst. **34**:557 (May) 1922.

Operative Procedure.—On December 4, Dr. Henry Wigderson operated, with the use of local anesthesia. A U-shaped incision was made in the skin within about 1 cm. of the tumor. The base of the cutaneous flap was in the supraorbital region. The flap was dissected and stripped rather freely. The tumor remained covered with periosteum. An incision was then made through the periosteum down to the bone in the region of the margin of the bony defect along its superior border. In this way the tumor was dissected free from the bone, and the typical smoothly scalloped edge of the bony deformity produced by the epidermoid

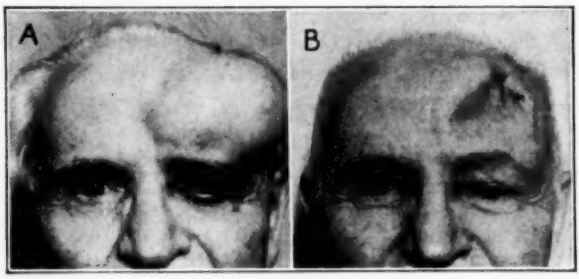


Fig. 1.—*A*, preoperative appearance of the patient, showing an epidermoid producing closure of the lid; *B*, postoperative appearance.

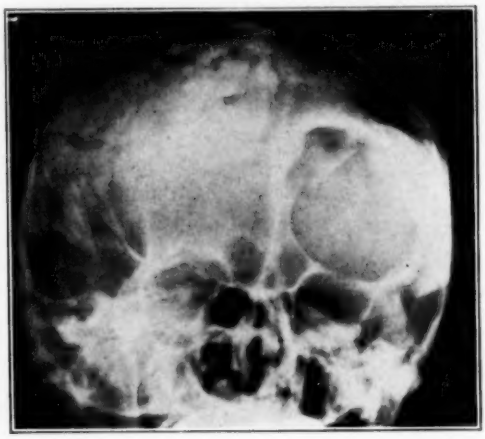


Fig. 2.—Typical roentgenogram, showing loss of bone in the frontal area, as well as in the orbit.

was revealed. The tumor was dissected away from the edge of the bone for a distance of about 5 to 6 cm. At this stage the capsule was torn. An effort was made to maintain the tumor in a fairly intact state and to continue the dissection, but this attempt was unsuccessful. The capsule was then incised widely, and the contents, consisting chiefly of 75 cc. of caseous-looking material and a yellowish, oily fluid, evacuated. The upper part of the capsule was cut away. The capsule remained adherent to the underlying dura. Some of this was stripped free, but in order that the dura might not be torn in the process, and in view of

the age of the patient, it was not attempted to remove the capsule completely. After the tumor had been removed, the bony deformity was observed as follows: There was a defect measuring approximately 7 cm. in diameter in the left frontal region. In the superolateral two thirds the bony edge was beveled. In the medial third the bone was undermined and somewhat elevated. The pulsating dura was observed at the base of the tumor cavity. Anteriorly, the supraorbital ridge and the anterior two thirds of the roof of the orbit had been destroyed (fig. 3). When the patient was asked to look up, the fascial roof of the orbit was retracted, and when he was asked to look down, it moved upward. A Penrose drain was brought out through a stab wound posteriorly. The skin was closed with a continuous modified Steward stitch. A large bony defect and redundancy of skin remained. The postoperative course was uneventful.

Histologic Study.—The lining of the wall of the cyst showed epithelial cells suggestive of the stratum granulosum (fig. 4). The cystic fluid contained cholesterol crystals.

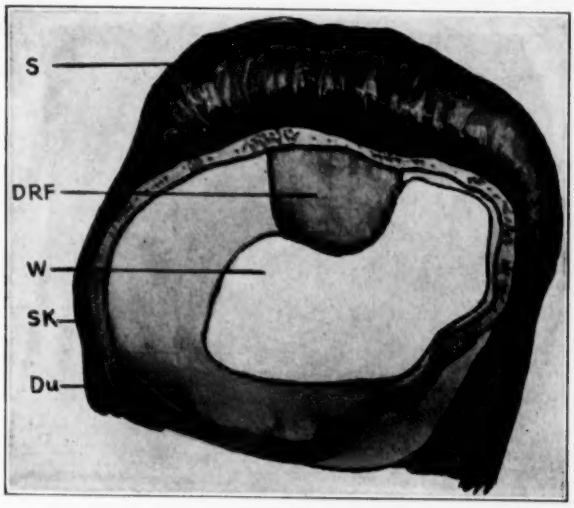


Fig. 3.—Appearance of the operative field at the conclusion of the operation. Here, *S* indicates scalp; *DRF*, defect in roof of orbit; *W*, wall of cyst; *SK*, defect in skull, and *DU*, dura.

Postoperative Course.—Reexamination on Feb. 17, 1947 disclosed that the palpebral apertures were of equal width (8 mm.) (fig. 1 *B*). Corneal hyposensitivity was present in the left eye. No limitation of elevation of the globe was present, and the return of function was complete. With the stethoscope over the eyeball, normal breath sounds were heard over the right eye, but sonorous breath sounds were heard over the closed left eyeball. With the exophthalmometer at 103 mm., the right eye measured 16 mm. and the left eye 14 mm. In the cover test, exophoria for 13 inches (33 cm.) measured 2 Δ, and fusion was present.

COMMENT

Although Cushing advised complete removal of the wall of the cyst in order to prevent recurrence, the slow growth of the tumor in the

present case together with the adherence to the dura and the age of the patient, justified conservatism in surgical procedure. Cairns[5] reported 2 cases in which operation had been performed over seven years before, with only partial removal of the cyst wall, and no sign of recurrence had appeared.

The movement of the fascial roof of the orbit as observed on the operating table should be given some thought, so that the normal physi-

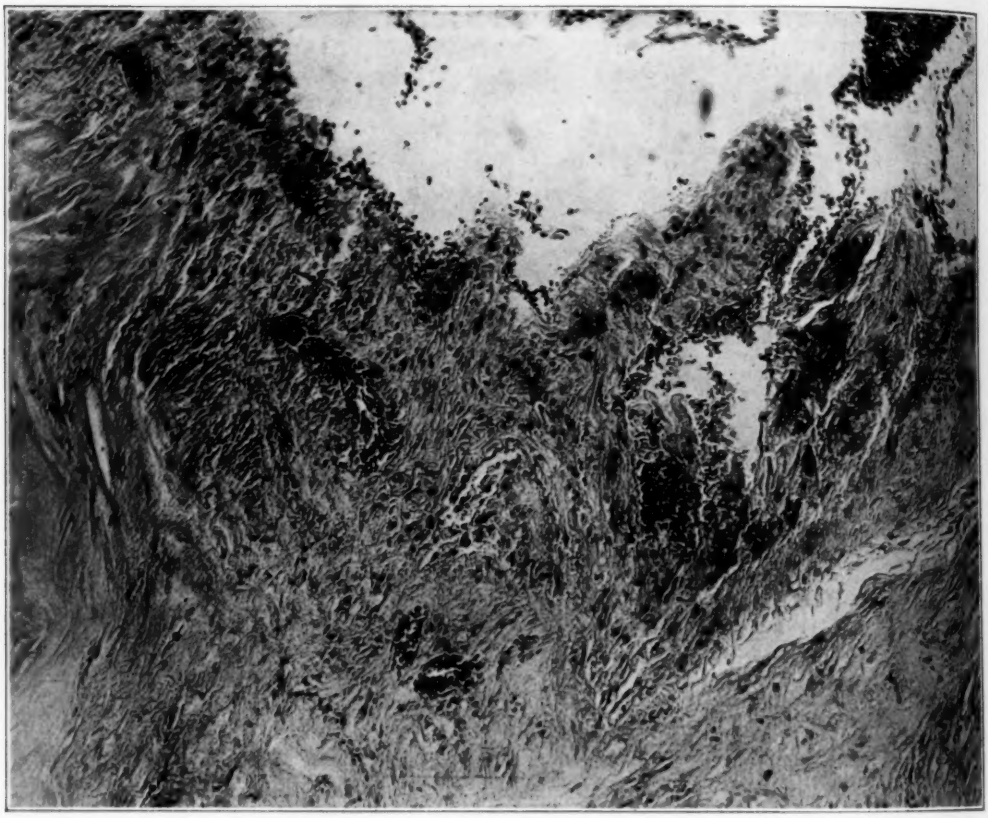


Fig. 4.—Photomicrograph, showing the epithelial cells lining the wall of the cyst. × 140.

ology of orbital movements as they are understood at present may be confirmed or corrected.

108 East Sixty-Sixth Street (21).

5. Cairns, H.: Ultimate Results of Operations for Intracranial Tumors: Study of Series of Cases After Nine Year Interval, Yale J. Biol. & Med. **8**:421 (May) 1936.

NORMAL OPTIC NERVE

I. Classification of the Optic Disk Based on Branching of the Central Retinal Artery

EVERET H. WOOD, M.D.
AUBURN, N. Y.

IT HAS long been obvious that inadequate attention has been paid to the detailed topography of the eyegrounds. The older works called attention to general relations, and Goldstein [1] even suggested that photography of the fundus might be useful in identification of criminals. Jensen [2] studied distribution of vessels in the fundus in general. In a recent paper, Cordes [3] reviewed the congenital and acquired anomalies of the optic disk but did not make a detailed study of the distribution of vessels on the disk itself. Opportunity was recently afforded me to study the normal topography of the eyegrounds in several hundred men in the armed forces. The first phase of this study was limited to the relation of the retinal arteries to the quadrants of the optic disk. A planned study of this limited scope seemed particularly worth while because of the relations of these root vessels to changes in the nerve and vessels associated with glaucoma; to swelling of the optic nerve, a possible complication of arteriosclerosis, and to a number of other conditions which need not be recited.

After examination of hundreds of normal eyegrounds, it became apparent that there are several definite and easily recognizable variations in the distribution of the vessels as they emerge from the optic nerve head. It is interesting to note the position of the vessels as they appear in the normal eye. The present paper is concerned with a simple classification of the nerve head based on the branching of the central artery of the retina.

The classification is made by dividing the surface of the nerve head into four quadrants, i. e., the upper and lower nasal and the upper and lower temporal. Since there are four main branches of the retinal artery, namely, the superior and inferior temporal and the superior and inferior nasal, there are nine possible variations in the distribution of these

1. Simon, C., and Goldstein, I.: New Scientific Method of Identification, New York State J. Med. **35**:901-906 (Sept. 15) 1935.

2. Jensen, V. A., in Kershner, C. M.: Blood Supply of the Visual Pathway, Boston, Meador Publishing Company, 1943, pp. 64-74.

3. Cordes, F. C.: Congenital and Acquired Anomalies of the Optic Disk, Arch. Ophth. **23**:1063-1089 (May) 1940.

vessels over the surface of the optic nerve head. These variations comprise the first nine classes. There is also a tenth class, in which the vessel does not follow the quadrant distribution. The distribution of the veins is disregarded in the makeup of this classification. The ten classes are described as follows:

Class 1. Nasal
All branches on nasal side of the disk.

Class 2. Nasal Oblique
Superior vessels in upper nasal quadrant; inferior vessels in lower temporal quadrant.

Class 3. Temporal Oblique
Superior branches in upper temporal quadrant; inferior branches in lower nasal quadrant.

Class 4. Temporal
All branches on temporal side of disk.

Class 5. Nasal—Temporal Up
Inferior branches in lower nasal quadrant; superior nasal branches in upper nasal quadrant; superior temporal branches in upper temporal quadrant.

Class 6. Nasal—Temporal Down
Superior branches in upper nasal quadrant; inferior nasal branches in lower nasal quadrant; inferior temporal branches in lower temporal quadrant.

Class 7. Temporal—Nasal Up
Inferior branches in lower temporal quadrant; superior nasal branches in upper nasal quadrant; superior temporal branches in upper temporal quadrant.

Class 8. Temporal—Nasal Down
Superior branches in upper temporal quadrant; inferior nasal branches in lower nasal quadrant; inferior temporal branches in lower temporal quadrant.

Class 9. Symmetric
One branch in each quadrant.

Class 10. Miscellaneous

VESSELS OF INDETERMINATE QUADRANT DISTRIBUTION

The nomenclature of the ten classes of optic nerve heads was based on the five general types which appear: (1) the symmetric group, in which each quadrant contains a branch; (2) the nasal groups, in which all vessels are on the nasal side; (3) the temporal groups, in which all vessels are on the temporal side; (4) the oblique groups, in which the superior portion of the axis provides the name—for example, in class 3 (the temporal oblique), the superior branches appear in the upper temporal quadrant and the inferior branches in the lower nasal quadrant, and (5) the miscellaneous group. Classes 5 through 8 do not adhere to the strict types of vascular distribution and are named for the dominating type, with the second component in the name indicating the

branch which does not belong to the main type. For example, in class 5 the nerve head has branches in both nasal quadrants; hence, it is named nasal, and it also has a branch in the upper temporal quadrant which deviates from the nasal type; so the class is named nasal—temporal up. The miscellaneous group does not fit into any of the aforementioned types.

PROCEDURE

The eyegrounds of every person who came to the ophthalmic clinic of a large Army general hospital were examined routinely for pathologic changes prior to refraction. During this examination the classification of the disk was determined by simple inspection of the disk. In most instances there was no difficulty whatever in determining the classification of the disk, at least for the first nine classes. In cases in which there was a question as to whether or not a branch was in the nasal or in the temporal quadrant because it crossed the disk near the division, a streak of light from an ophthalmoscope was directed to the vertical diameter of the

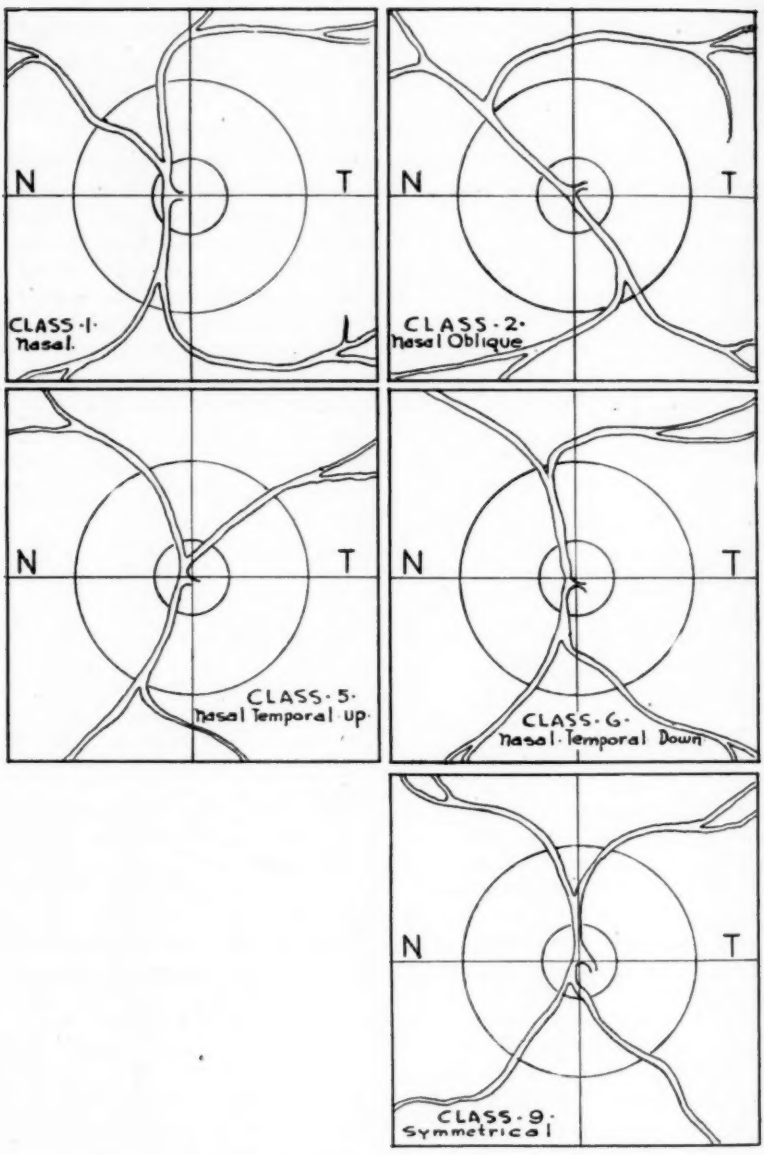
TABLE 1.—*Classification of 500 Optic Disks by the Distribution of Arterial Branches*

Class	First 200 Eyes		Second 200 Eyes		Last 100 Eyes		Total	
	No.	%	No.	%	No.	%	No.	%
1. Nasal	30	15	34	17	19	19	83	16.6
2. Nasal oblique	0	0	0	0	1	1.0	1	0.2
3. Temporal oblique	0	0	1	0.5	0	0	1	0.2
4. Temporal	0	0	0	0	0	0	0	0
5. Nasal—temporal up	73	36.5	71	35.5	32	32	176	35.2
6. Nasal—temporal down	16	8	18	9	12	12	46	9.2
7. Temporal—nasal up	0	0	2	1	1	1	3	0.6
8. Temporal—nasal down	0	0	0	0	0	0	0	0
9. Symmetric	71	35.5	63	31.5	26	26	160	32
10. Miscellaneous	10	5	11	5.5	9	9	30	6
Total	200	100	200	100.0	100	100	500	100

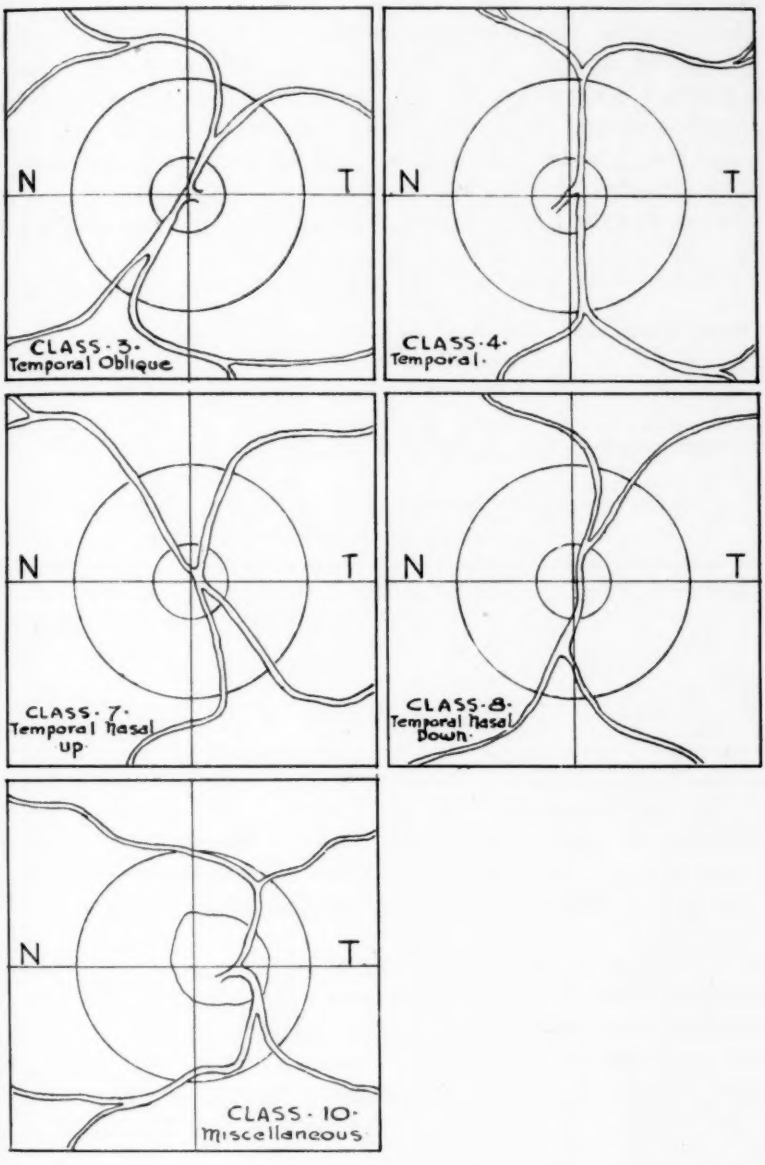
disk and a more careful observation made as to the exact class in which a particular disk should be placed. There was seldom any difficulty with regard to the upper and lower halves. More difficulty arose in distinguishing between the first nine classes and the so-called miscellaneous class, or class 10. It became very nearly a matter of personal judgment as to whether some disks should be placed in class 9 or class 10. Disks which had a large physiologic cup far to the temporal side, with vessels circling the edge of the disk, rather than branching on its surface, were those considered to be in class 10. As will be seen from the statistics which follow, such disks are not uncommon in persons with normal eyes, since approximately 10 per cent of the eyes studied had disks which appear in this class. It will be noted later that Sergeant Kestenbaum found a larger percentage of disks in class 10 than I did. In my opinion, the reason for this discrepancy is that he was not careful enough in distinguishing the exact difference between the first nine classes and the miscellaneous class, or class 10.

STATISTICAL ANALYSIS

A statistical analysis of 500 eyes, based on this classification, is shown in table 1. It will be noted that the classes in which the nasal



Normal optic nerve head, showing ten types of distribution of the branches of the central retinal artery.


N
T
CLASS·3·
Temporal Oblique
N
T
CLASS·4·
Temporal·
N
T
CLASS·7·
Temporal Nasal
up·
N
T
CLASS·8·
Temporal Nasal
Down·
N
T
CLASS·10·
Miscellaneous

quadrants were devoid of main vascular branches were greatly in the minority. In other words, almost invariably there were vessels and their branches on the nasal side of the optic disk. Frequently, either the lower or the upper temporal quadrant was devoid of main branches of the retinal arteries. It is interesting to note that in this series of 500 eyes not a single one was placed in class 4, in which the vessels are on the temporal side of the disk, or a single one in class 8, in which the superior branches are in the upper temporal quadrant and the inferior branches in the nasal and temporal quadrants, respectively. Six per cent of the 500 eyes were placed in class 10, in which the optic

TABLE 2.—*Tabulation of Individual Characteristics of the Optic Disks of 250 Subjects*

	First 100 Eyes		Second 100 Eyes		Last 60 Eyes		Total	
	No.	%	No.	%	No.	%	No.	%
Eyes both in same class	53	53	39	39	12	24	104	41.6
Eyes in different classes	47	47	61	61	38	76	146	58.4
Total	100	100	100	100	50	100	250	100.0

TABLE 3.—*Classification of 324 Optic Disks by Distribution of Arterial Branches**

Class	200 Eyes		124 Eyes		Total No. of Eyes (324)	
	No.	%	No.	%	No.	%
1. Nasal	75	37.5	33	26.6	108	33.3
2. Nasal oblique	1	0.5	0	0	1	0.3
3. Temporal oblique	1	0.5	1	0.8	2	0.6
4. Temporal	0	0.0	0	0	0	0.0
5. Nasal—temporal up	24	12.0	16	12.9	40	12.2
6. Nasal—temporal down	19	9.5	8	6.5	27	8.3
7. Temporal—nasal up	4	2.0	2	1.6	6	1.8
8. Temporal—nasal down	2	1.0	1	0.8	3	0.9
9. Symmetric	19	9.5	6	4.8	25	7.6
10. Miscellaneous	55	27.5	57	46.0	112	35.0
Total	200	100.0	124	100.0	324	100.0

* The statistical data in this series were obtained by Sgt. William Kestenbaum.

cup is very large, in many instances quite deep and much farther toward the temporal side of the disk than the cups in the other classes.

Another interesting feature of the analysis is that 16.6 per cent, or 83, of the 500 eyes fell in class 1, in which all the branches appear on the nasal side of the disk; surprisingly, only 160 of 500 eyes, or 32 per cent, fell in class 9, in which the branches are distributed in all four quadrants. The next most frequent occurrence was class 5, in which the lower nasal and the two upper quadrants contain the main branches of the vessels.

Table 2 presents a comparison of the two eyes of the 250 subjects which made up the statistical data for table 1. There were 104 subjects,

or 41.6 per cent, whose optic disks fell in the same class and 146 subjects, or 58.4 per cent, whose optic disks fell in different classes.

Having obtained these data, I was not satisfied to use my own observations only in arriving at statistical conclusions. I had Sgt. William Kestenbaum, an optometrist, with whom I was associated in the ophthalmic department of an Army general hospital, make a similar study. His series comprised 324 eyes, or 162 subjects. His results, though not exactly the same as mine, bear out the general distribution in a remarkably similar fashion. Tables 3 and 4 present his figures. The most interesting feature of his observations is that the disks of 72, or 44 per cent, of his patients fell in the same class and the disks of 90, or 56 per cent, in different classes.

Two other observations were made during this study. 1. The retinal artery emerges from the disk on the nasal side of the retinal vein. To date, I have seen only 1 case in which the retinal artery emerged temporal to the vein. 2. It is possible to recognize the vascular pattern and to classify the optic disk on this basis as long as

TABLE 4.—*Tabulation of Individual Characteristics of Optic Disks in 162 Subjects* *

	100 Eyes		62 Eyes		Total	
	No.	%	No.	%	No.	%
Both eyes in same class.....	49	49	23	37.1	72	44
Eyes in different classes.....	51	51	39	62.9	90	56
Total.....	100	100	62	100	162	100

* The series was studied by Sgt. William Kestenbaum.

three hours after death. I had several opportunities to observe the eyegrounds immediately after death and to check them at frequent intervals up to the time when the arterial pattern was no longer visible because of postmortem changes. In none of the eyegrounds examined could the arterial distribution be seen after the three hour period.

CONCLUSIONS

1. The normal optic disk may be classified into ten distinct types, based on the distribution of the main branches of the central retinal artery.

2. It is extremely rare to find either the upper or the lower nasal quadrant devoid of main branches of the central retinal artery.

3. Up to the time of writing, no case has been found in which the whole nasal half of the disk was devoid of the main branches of the central retinal artery.

4. Less than one-half the 412 subjects studied had optic disks which were in the same class.

5. The arterial pattern of the eyegrounds can be recognized at least up to three hours after death.

6. The central artery of the retina emerges from the disk on the nasal side of the central vein in a large majority of all eyes.

Dr. John N. Evans rendered valuable assistance in the preparation of this paper; Sgt. William Kestenbaum gathered the statistical material, and Mr. Robert S. Richards gave technical assistance in the composition of the diagrammatic sketches illustrating the paper.

120 Genesee Street.

EFFECT OF ROENTGEN THERAPY ON EXPERIMENTAL OCULAR VACCINIA IN NONIMMUNE AND IN PARTIALLY IMMUNE RABBITS

GEORGE T. HARRELL, M.D.

CHARLES H. REID, M.D.

J. MAXWELL LITTLE, Ph.D.

JAMES W. MANKIN, M.D.

HAL W. PITTMAN, M.D.

LAWRENCE BYERLY HOLT, M.D.
AND
LESLIE M. MORRIS, M.D.
WINSTON-SALEM, N. C.

ACCIDENTAL ocular infection with vaccinia occurs usually as a complication of prophylactic immunization against smallpox. Rarely infection results from a laboratory accident during the preparation or experimental use of the virus. The danger of ocular infection lies in involvement of the cornea, with subsequent development of an opaque scar.

The prevention of permanent impairment of vision is a goal in treating any infection of the eye. The therapy of ocular vaccinia is not standardized. Various forms of radiation therapy have been used in human beings and in rabbits. The present experiments were suggested by the beneficial results obtained from the use of roentgen therapy for an accidental ocular infection in a partially immune human being.[1]

EXPERIMENTAL STUDY

Rabbits were used to study the effect of direct irradiation at different stages of ocular vaccinia in nonimmune and partially immune animals and the nonspecific effect of irradiation of a distant primary lesion.

MATERIALS AND METHODS

Glycerinated calf lymph virus containing brilliant green was obtained from the North Carolina State Department of Health. All the virus used in the preliminary

From the Departments of Internal Medicine, Physiology and Pharmacology, and Radiology, Bowman Gray School of Medicine of Wake Forest College and the North Carolina Baptist Hospital.

1. Pittman, H. W.; Holt, L. B., and Harrell, G. T.: The Effect of Irradiation, Immunity and Other Factors on Vaccinial Infection: A Review Illustrated by the Report of a Secondary Ocular Infection Treated with Roentgen Rays, Arch. Int. Med. **80**:61-67 (July) 1947.

experiments was from the same lot, and that used in subsequent experiments was from another lot; the virus was stored in sealed capillary tubes in a refrigerator at 8 C.

One hundred and six rabbits, of both sexes, were used. The majority of the rabbits were from 2 to 4 months old, but a few rabbits in the control groups were older. All the females were kept in individual cages; some of the males in later experiments were grouped together. All animals were maintained on a stock diet ("Kasco complete rabbit ration," Kasco Mills, Inc., Toledo, Ohio) for at least eight days before the experiments were started. The animals were fed and watered at regular intervals, usually twice daily, to insure adequate nutrition and hydration.

Ocular anesthesia was obtained by instilling in the conjunctival sac two drops of a 2 per cent aqueous solution of butacaine sulfate U. S. P. Fifteen minutes later three superficial vertical scratches were made on the anesthetized cornea with a sterile sewing needle. One drop of virus was placed in the conjunctival sac, and the lid was closed, opened and reclosed in order to spread the virus over the entire cornea. The eyelid was always handled with the greatest care to avoid abrasion of the palpebral conjunctiva. Only one eye was inoculated in each rabbit.

The left flank of each rabbit which received inoculation in the skin was cleared of hair with animal clippers. Inoculation was performed by the combined technic of linear scratches and multiple punctures of the skin.

In preliminary experiments, roentgen therapy was administered with the following factors: 100 kilovolts; 5 milliamperes; distance 20 cm.; portal diameter 2.5 cm; no filter except the 0.5 mm. of aluminum inherent in the tube; exposure, thirty seconds. The half value layer equaled 0.80 mm. of aluminum. All animals except those in group A were treated three times, and thus received 102 r in air per treatment; because of technical difficulties the rabbits in group A received only two treatments. In subsequent experiments the factors were slightly altered, so that 120 kilovolts at a distance of 21 cm. was used; the half value layer then equaled 0.94 mm. of aluminum, and the animals received 105 r in air at each treatment.

The eyes of all animals in the preliminary (qualitative) experiments were examined daily for lacrimation, exudate, vascular engorgement, edema, corneal ulcers and opacities, miosis, blepharitis, iritis and pannus; the flanks were examined for edema, erythema, macules, papules, vesicles and pustules. The reactions were graded on a scale of 1 to 6 plus. The animals were observed daily for twenty-eight days, and then at irregular intervals for fifty-three days in experiment 1 and for thirty-six days in experiment 2. In subsequent (quantitative) experiments (3A and 3B) the animals were observed for sixteen days, and only for opacity of the cornea. The degree of corneal opacity was graded at the time the animals were killed.

Observation of the extent of the ocular lesion is a rough estimation of damage, but gives no information on the degree of impairment of vision. In order to measure objectively the decrease in transmission of visible light in the cornea of an experimental animal, it was necessary to devise a quantitative method. A technic employing the photoelectric colorimeter was developed.[2] In experiment 3 the corneas of the infected and uninfected eyes of each animal were removed at the time the rabbit was killed. The control animals in group L were killed on the twenty-first day, and beginning on the twenty-fifth day animals were killed in multiples of five, 1 animal from each of the five test groups (J, K, M, N and O).

2. Little, J. M.; Mankin, J. W.; Reid, C. H., and Harrell, G. T.: The Scatter and Absorption of Light by the Excised Cornea at Various Wavelengths Determined by a Photoelectric Method, J. Clin. Investigation **26**:416 (May) 1947.

PRELIMINARY EXPERIMENTS

Experiment 1: Determination of the Optimum Time for Roentgen Therapy of Primary Vaccinia of the Cornea in Nonimmune Rabbits.—Four groups consisting of 3 animals each, and another consisting of 2 animals, were used. In group A the treatments were given twenty-four and forty-eight hours after inoculation (as soon as secondary inoculation in a patient would be suspected, and before any lesion would have appeared); in group B the treatments were started at the time of appearance of the earliest visible lesion (vesiculation) and were given sixty-six, seventy-eight and ninety hours after inoculation; the rabbits in group C received treatment at the height of the lesions—one-hundred-sixty-eight, one-hundred-eighty and one-hundred-ninety-two hours after inoculation. The corresponding times for therapy will not necessarily occur at the same intervals after inoculation in human subjects, since vaccinia runs a more rapid course in rabbits than in human beings.

The rabbits in group D received no radiation and served as a control on the effect of roentgen therapy. Group E, consisting of 2 rabbits, served as a control on the extent of damage done by trauma, irradiation and chemical irritation. The corneas of both animals were anesthetized, but no vaccine was applied. The cornea of one animal was irradiated but not scarified, and that of the other was scarified but not irradiated.

Results: All rabbits in groups A, B, C and D showed violent inflammatory reactions of the cornea and bulbar conjunctiva, beginning on the third day after inoculation and reaching a peak in the second week. The range in severity of the reactions in each group was moderately wide. The lesions subsided by the end of the third week, leaving in every instance an opacity of the cornea. In the rabbits of group A, irradiated on the first two days after inoculation, the lesions appeared one to one and one-half days later than in the controls (group D), but the peak of the reaction was reached at the same time. At the height of the process no appreciable difference between the irradiated lesions and those which had not been irradiated was detected. As the inflammatory process began to subside, between the ninth and the eleventh day, it became apparent that the acute lesions regressed more rapidly in the irradiated group (fig. 1) and that the severest chronic lesions occurred in the corneas of the animals which had not been irradiated (fig. 2). The slightest residual opacities were noted in group C, which received roentgen therapy at the height of the lesion. Figure 3 illustrates the best and the worst results in this group and in group D, in which the rabbits were untreated. The animals in groups A and B also showed less corneal opacity than those in group D. No visible lesions resulted from anesthetization, scarification or irradiation (group E). When the lesions at

twenty-eight days are scored from 1 to 6 plus, the difference between the means for the opacities of group C and D, or between the opacities of groups A, B, C together and those of group D gives a relative deviate of *t* which is statistically significant [3] (table 1).[4]

One rabbit in group A died on the thirteenth day of the experiment; 2 rabbits in group B died on the twenty-third and twenty-fifth days, respectively. The cause of death of the last 2 animals was not defi-

TABLE 1.—*Statistical Comparison of Sums of Scores of Opacities (Experiment 1)*

Days	Groups Compared	n	Calculated Value for t	Significant Value for t (5%)	Interpretation
7	ABC and D	10	1.11	2.228	Not Significant
	C and D	4	0.45	2.776	Not Significant
14	ABC and D	9	1.12	2.262	Not Significant
	C and D	4	0.54	2.776	Not Significant
21	ABC and D	9	1.12	2.262	Not Significant
	C and D	4	1.23	2.776	Not Significant
28	ABC and D	7	3.74	2.365	Significant
	C and D	4	3.43	2.776	Significant

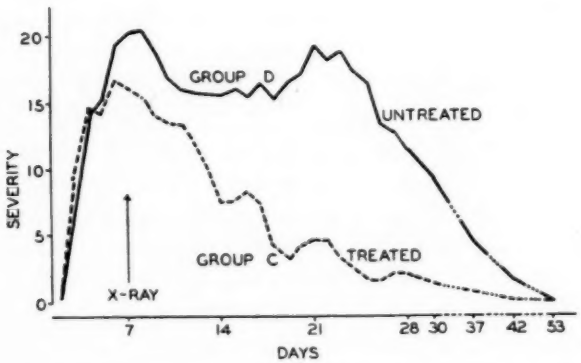


Fig. 1.—The mean of the sums of the scores of all acute lesions of the eye in group C (treated with roentgen radiation) and group D (untreated) are plotted as three point moving averages. The curves for groups A and B (also treated with roentgen radiation, but at slightly different intervals after inoculation) closely followed that for group C.

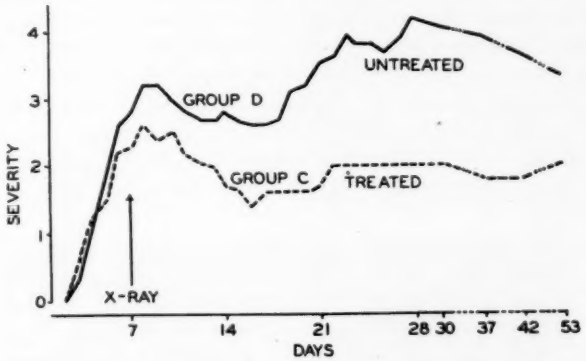
nitely determined, but in no instance was death thought to be due to generalized vaccinia or to an overdose of roentgen radiation.

Experiment 2.—Role of Partial Immunity in Ocular Vaccinia: A. Effect of Direct and Distant Roentgen Therapy on Secondary Vaccinia of the Cornea in Animals Partially Immune as a Result of Primary Inoculation of the Skin.—The flanks of 6 rabbits were inoculated with vaccinia virus; seventy-two hours later,

3. Dr. Nash Herndon assisted in the statistical criticism.

4. Snedecor, C. W.: Statistical Methods, Ames, Iowa, Iowa State College Press, 1940.

after the local lesions had appeared in the flanks, the corneas were inoculated with fresh virus. Group F, consisting of 3 rabbits, received roentgen treatments to the eye one hundred and thirty-six, one hundred and forty-eight and one hundred and sixty-eight hours, respectively, after inoculation of the flank (two and a



Gig. 2.—The mean of the sums of the scores of corneal opacities—the only chronic lesion—in group C (treated with roentgen radiation) and group D (untreated) are plotted as three point moving averages. The curves for groups A and B (also treated with roentgen radiation, but at slightly different intervals after inoculation) closely followed that of group C until twenty-one days after inoculation. They then gradually rose until the twenty-eighth day and thereafter remained parallel to the curve for group C.

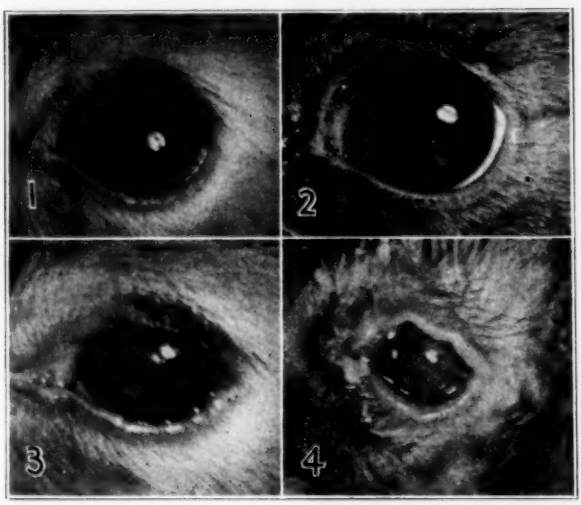


Fig. 3.—Primary ocular vaccinia in nonimmune rabbits thirty-one days after inoculation (experiment 1). *1* shows the best, and *3* the worst, results in group C (those treated with roentgen radiation at the height of the lesion—seven and eight days after inoculation); *2* shows the best, and *4* the worst, results in group D (untreated).

half, three and three and a half days after inoculation of the eye). In group G, consisting of 3 rabbits, the treatments were given at the same time, but to the flank rather than to the eye.

Results: The reactions to secondary inoculation of the cornea were less severe and the residual corneal opacities were less dense than those which followed primary inoculation (experiment 1). Severer reactions occurred in the group which received roentgen therapy to the flank (group G) than in the group in which the eyes were directly irradiated (group F). The results would indicate that the effect of direct irradiation of the corneal lesion is greater than the systemic effect of irradiation of a distant primary site. Local irradiation seemed to have less effect on the lesions of the flank than on the ocular lesions.

B. Effect of Roentgen Therapy on Primary Vaccinia of the Cornea in Animals Receiving Secondary Inoculations of the Flank.—The corneas of 6 rabbits were inoculated with vaccinia virus; after seventy-two hours, when the earliest lesions were visible, the flanks of the animals were inoculated with fresh virus. Group H,

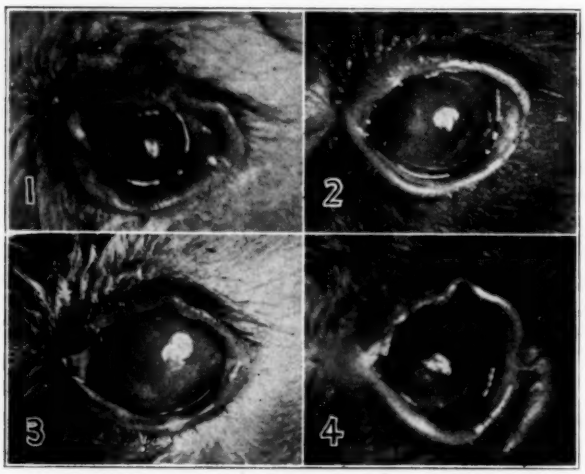


Fig. 4.—Effect of partial immunity on ocular vaccinia (experiment 2). The lesions shown are the severest in each of the four groups, thirteen days after ocular inoculation.

1 and *2* show the lesions of secondary ocular vaccinia. The eye in *1* (group F) was treated with roentgen radiation two and three days after inoculation of the eye and six and seven days after inoculation of the flank; the eye in *2* (group G) was treated at the same time, but with roentgen radiation to the flank rather than to the eye.

3 and *4* show the lesions of primary ocular vaccinia in animals receiving secondary inoculation of the flank seventy-two hours after inoculation of the eye. The eye in *3* (group H) was treated with roentgen radiation six days after inoculation of the eye; the eye in *4* (group I) was not treated.

consisting of 3 rabbits, received roentgen treatments to the eye one hundred and thirty-six, one hundred and forty-eight and one hundred and sixty hours after inoculation of the cornea. Group I, consisting of 3 rabbits, received no roentgen therapy and served as a control on the effect of immunity alone.

Results: The lesions in the group which received roentgen therapy to the eye after secondary inoculation of the flank (group H) were comparable in severity to those in group F (secondary vaccinia of the cornea treated by local irradiation). The severest reaction observed in experiment 2 was that in an animal in group I, which received no roentgen therapy (fig. 4). The acute lesions in the corneas of the irradiated animals subsided more rapidly than those in the nonirradiated ones.

Four animals in experiment 2 failed to show corneal lesions, though 1 of these had a conjunctival lesion. The 3 animals which failed to show ocular lesions were evenly distributed in groups F, H and I. The animals in experiment 2 were all slightly smaller and younger than those in experiment 1. The virus may have become attenuated during three weeks of storage between the two experiments, though it had not been removed from the refrigerator. That the virus was active is shown by the development of lesions on the flanks of all animals and by subsequent successful human inoculations. Difference in technic is a possible explanation of the failures in experiment 2, since the inoculations were done by a different member of the team and the scratches were sometimes slightly shorter than those in experiment 1. Partial immunity induced by inoculation of the skin may have been a greater factor than any of these. Because of the small size of the groups in which the failures occurred, the conclusions reached cannot be subjected to statistical analysis.

Conclusions.—On the basis of the preliminary experiments, it was concluded that roentgen therapy for primary or secondary vaccinial infection of the eye hastens regression of the acute lesion and probably diminishes the residual corneal opacities. The optimum time for therapy appears to be between the appearance of the lesion and the time it reaches its height. Treatment very early, as soon as inoculation would be suspected, delays the appearance, but does not prevent the full development, of the lesion.

ADDITIONAL EXPERIMENTS

The investigation was pursued further, with the number of variables reduced and the size of the individual groups increased. The corneal inoculations were done simultaneously in all groups to avoid possible attenuation of the virus. Measurements of the decrease in transmission of light were made within thirty minutes of the death of the animal.

Experiment 3.—Determination of the Relative Value of Roentgen Therapy and Partial Immunity in Corneal Vaccinia: A. Value of Roentgen Therapy in Primary Corneal Vaccinia.—In group J, consisting of 15 animals, the treatments were given one hundred and three, one hundred and twenty-seven and one hundred and forty-eight hours (four, five and six days) after inoculation. In group K, consisting of 15 rabbits, the animals received no radiation and served as positive controls on the effect of roentgen therapy. Group L, consisting of 5 rabbits, served as a control on the extent of damage done by trauma, irradiation and chemical irritation—negative control. Both eyes of each animal were anesthetized and scarified; only the right eye was irradiated.

Results: All animals in groups J and K showed inflammatory reactions of the corneas and bulbar conjunctivas; corneal opacities were noted in each. No visible lesions were seen in any animal in group L. The galvanometric readings of the photoelectric colorimeter (T) were subtracted from 100 to give the per cent of loss of transmission by light scattered and absorbed; these data are recorded in table 2. Since the decrease in transmission of light by the cornea of the right (irradiated) and that by the cornea of the left (nonirradiated) eyes in group L were almost identical, the data on both eyes are recorded to increase the size of the sample for statistical analysis.

B. Value of Roentgen Therapy and Partial Immunity in Secondary Vaccinia of the Cornea in Animals Partially Immune As a Result of Primary Inoculation of the Skin.—Thirty rabbits were inoculated on the flank with vaccinia virus three days before inoculation of the corneas. The corneas of groups J and K

(experiment 3A) and groups M, N and O were inoculated at the same time from the same lot of virus, so that results for the various groups could be more accurately compared. Group M, consisting of 15 rabbits, received roentgen treatments to the eye on the seventh, eighth and ninth days after inoculation of the flank (one hundred and three, one hundred and twenty-seven and one hundred and forty-eight hours after inoculation of the eye). Group N, consisting of 15 rabbits, received no radiation and served as a control on the effect of partial immunity alone. Group O, consisting of 15 rabbits, was inoculated on the flank three days after inoculation of the eye, but received no radiation therapy and served as a further control on the participation of the eye in immunity.[5]

TABLE 2.—*Decrease in Transmission of Light in Eyes with Primary Vaccinia Treated with Roentgen Radiation**

	100—T† Filter 660			100—T Filter 540			100—T Filter 440		
Group	J *	K *	L *	J	K	L	J	K	L
1	4.75	5.50	1.75	7.00	10.25	4.00	16.00	46.00	6.50
2	47.75	2.75	1.25	60.00	4.00	3.00	13.00	18.00	7.00
3	2.50	3.75	0.75	5.00	6.00	2.50	20.00	8.00	11.25
4	6.50	23.75	1.75	10.00	32.00	3.50	7.00	6.00	.....
5	6.50	7.25	1.00	8.00	11.00	2.50	4.50	11.50	.....
6	5.25	0.50	1.50	11.25	3.75	3.75	6.50	10.50	.....
7	3.00	1.75	1.50	3.50	3.25	3.50	19.50	14.50	.....
8	0.75	3.50	0.75	1.50	6.50	3.50	9.50	8.00	.....
9	1.50	4.25	1.25	3.25	6.50	2.50	6.50	10.00	.....
10	8.50	5.00	0.75	13.00	8.00	2.75	8.00	12.00	.....
11	3.25	3.75	....	5.25	4.50	....	10.50	9.00	.....
12	2.25	4.25	....	3.50	6.00	....	7.50	6.00	.....
13	2.75	5.50	....	4.50	7.50	....	.....	.....	.....
14	4.50	3.50	....	6.25	5.25	....	.....	.....	.....
15	4.00	1.00	....	5.00	2.25	....	.....	.....	.....
Mean	6.91	5.06	1.22	9.80	7.78	3.15	10.70	13.29	8.25
N	15	15	10	15	15	10	12	12	3
σ	11.485	5.462	0.339	14.200	7.130	0.568	5.255	10.862	2.13

* Group J was not treated (positive control); group K was treated; group L was not inoculated (negative control).

† T represents the galvanometric readings of the photoelectric colorimeter.

Results: All animals in groups M, N and O showed inflammatory reactions of the corneas and bulbar conjunctivas. Corneal opacities were not present in 2 animals of group M and in 3 animals of group N; on the sixteenth day, 2 animals of group O showed no corneal opacities, and the corneas of 4 additional animals cleared, so that no visible lesions were present in 6 rabbits at the time the animals were killed.

5. Groups P and Q, on which data are given in the paper describing a photoelectric method for measurement of transmission of light, will not be further considered here.[2] The data designated as group P represent readings on the untraumatized and uninoculated cornea of rabbits with ocular vaccinia selected at random from groups J, K, M, N and O. These data indicate the degree of impairment of transmission of light by possible spread of the infection by natural routes to the opposite eye of the animal. Group Q consisted of 5 normal rabbits.

Pustules did not develop in the flanks of 10 animals in group M; only 5 animals which had evidence of severe infection of the flank survived for the duration of the experiment; hence, this group was quite small. Only 1 animal in group N failed to have a take on the flank; this animal had a grossly infected left (uninoculated) eye. All animals in group O had satisfactory takes on the flank.

Four animals in group M died after the fourteenth day of inoculation of the eye, and before they were killed; 1 animal in group N died on the day after inoculation of the eye. The animal from group N showed bilateral abscesses of the lung with empyema at autopsy; no apparent cause of death was observed in the others.

TABLE 3.—*Decrease in Transmission of Light in Eyes with Secondary Vaccinia Treated with Roentgen Radiation**

	100—T Filter 660			100—T Filter 540			100—T Filter 440		
Group	M *	N *	O *	M	N	O	M	N	O
1	3.75	1.50	3.25	6.00	3.50	5.50	11.00	6.00	14.00
2	....	1.00	6.75	3.00	3.50	9.75	11.50	6.00	26.50
3	2.75	2.00	2.00	4.25	3.75	4.25	7.50	11.00	7.00
4	8.75	1.75	3.00	12.25	3.50	5.25	20.00	7.00	8.50
5	6.75	1.50	13.75	10.75	5.50	18.00	.....	4.50	17.00
6	4.50†	1.25	0.75	6.25†	3.00	3.25	30.50†	32.00	24.00
7	3.00†	0	3.00	5.50†	1.50	3.50	7.00†	6.00	6.00
8	63.25†	9.75	7.50	75 50†	13.75	10.00	7.00†	39.50	7.00
9	13.75†	2.00	11.25	19.50†	3.00	16.00	.....	8.00	4.50
10	0.50†	21.75	2.50	3.75†	29.00	4.00	.....	8.00	14.00
11	3.25†	2.50	2.00	3.50†	4.25	3.75	.....	8.00	9.00
12	....	3.75	1.25	....	4.75	2.50	.....	.....	10.00
13	....	2.75	6.00	....	4.25	8.75	.....	.....	.....
14	....	....	3.50	....	....	5.50	.....	.....	.....
15	....	....	3.50	....	....	6.00	.....	.....	.....
Mean	5.50 (10.97)	3.92	4.66	7.25 (13.65)	6.40	7.06	12.50 (13.50)	12.36	12.27
Number	4 (10)	13	15	5 (11)	13	15	4 (7)	11	12
σ	2.39 (18.739)	5.846	3.744	3.62 (21.101)	7.399	4.656	4.59 (8.775)	11.802	7.095

* Group M was treated; group N was not treated (immune control); group O was not treated and received inoculation of flank after inoculation of eye.

† These animals did not get pustular takes on the flanks; the calculations within parentheses () include these data.

The degree of impairment in transmission of light is recorded in table 3.

Statistical Analysis.—The comparison of the effect of roentgen therapy and immunity on the transmission of light is given in table 4. The values for t was calculated by "Student's" method.[4] The impairment of transmission of light as determined by the photoelectric technic apparently was not significantly altered by either roentgen therapy or immunity or by a combination of the two. Though the number of animals in group M was reduced by the failure of pustules to develop in all after inoculation of the flank, the interpretation of the statistical analysis was not altered by recalculation of the data after the inclusion of animals without pustular takes.

COMMENT

The experiments do not clearly demonstrate that either immunity or roentgen therapy protects the eye against permanent damage by vaccinial infection. Opti-

TABLE 4.—*Statistical Comparison of Effects of Roentg en Therapy and Immunity on Transmission of Light* *

Groups Compared	Effect Evaluated	Filter 660				Filter 540				Filter 440			
		n	Calculated t	Significant Level (5%) t	Interpretation	n	Calculated t	Significant Level (5%) t	Interpretation	n	Calculated t	Significant Level (5%) t	Interpretation
K and J	Effect of roentgen therapy on primary vaccinia	28	0.564	2.048	Not Significant	28	0.492	2.048	Not Significant	22	0.925	2.074	Not Significant
M and N	Effect of roentgen therapy on secondary vaccinia	15 (21)	0.5146 (1.28)	2.131 (2.080)	Not Significant	16 (22)	0.2417 (1.62)	2.120 (2.074)	Not Significant	13 (16)	0.0225 (0.219)	2.160 (2.120)	Not Significant
M and J	Effect of immunity on roentgen therapy	17 (21)	0.2389 (0.607)	2.110 (2.080)	Not Significant	18 (24)	0.2370 (0.554)	2.101 (2.064)	Not Significant	14 (17)	0.2777 (1.075)	2.145 (2.110)	Not Significant
M and K	Effect of immunity plus roentgen therapy	17 (23)	0.1619 (1.161)	2.110 (2.069)	Not Significant	18 (24)	0.1616 (1.009)	2.101 (2.064)	Not Significant	14 (17)	0.1241 (0.043)	2.145 (2.110)	Not Significant
K and N	Effect of partial immunity	13	0.532	2.056	Not Significant	13	0.502	2.056	Not Significant	11	0.197	2.080	Not Significant
O and K	Effect of secondary inoculation of the flank	28	0.234	2.048	Not Significant	28	0.326	2.048	Not Significant	22	0.272	2.074	Not Significant
O and N	Participation of the eye in immunity	26	0.408	2.056	Not Significant	26	0.286	2.056	Not Significant	21	0.022	2.080	Not Significant

* The figures in parenthesis were obtained by including data on animals in group M which did not get a take on the flank.

mum conditions to insure infection were met; the inoculum was large; diet, vitamin and fluid intakes were maintained, and the possibility of pseudoestrus was avoided.[1]

Value of Roentgen Therapy.—Roentgen therapy appeared to be of value in the treatment of acute lesions of ocular vaccinia, as judged by clinical observation. The clinical impression that roentgen therapy may be valuable in decreasing residual corneal opacities was not borne out by experiments on the transmission of light, utilizing a photoelectric technic. Roentgen therapy would appear to have the same value in the treatment of secondary as of primary vaccinia; immunity did not appear to enhance the value of roentgen therapy for either acute or chronic lesions. The values for the means of the decrease in transmission of light might suggest that roentgen therapy was harmful; this is not borne out by the statistical comparison. The differences in transmission between the corneas of uninfected but traumatized animals which were irradiated and corneas which were not irradiated (right and left eyes of group L) and between the corneas of groups J and K and the corneas of groups M and N were so small that it was concluded that roentgen therapy was not harmful and did not increase corneal opacities.

Role of Immunity in Ocular Vaccinia.—Clinical observation on patients and experimental animals has convinced us that partial immunity protects against residual corneal opacities. If the mean of the decrease in transmission of light in group M as calculated with the animals included in which pustular takes of the flank did not occur is compared with the mean calculated with these animals excluded, it is evident that the decrease was less in the animals which should have partial immunity from the pustular take. The fact that takes were obtained on the flank of the animals of group O after inoculation of the eye and that takes were obtained in the eye of the rabbits of group N after inoculation of the flank could be interpreted to indicate that the eye does not participate in immunity. Cellular immunity may develop in the eye, but more slowly than in the skin, and a sufficient interval may not have elapsed after inoculation of the flank for the eye to have become partially immune; the experiments reported cannot answer this question.

On the basis of the results in group O, secondary inoculation of the arm in a human being with ocular vaccinia may be of no value but would appear to do no harm.

The lack of statistical significance in the comparison of group M with other groups, whether calculated to include or to exclude the animals without pustular takes on the flank, could be interpreted to indicate that partial immunity prevents little damage which leads to decrease in transmission of light. These data could also be interpreted to indicate that a low degree of immunity, resulting from a presumptive take in the flank, which showed edema and erythema but no pustules, affords the same protection as a violent local take.

Critique of the Photoelectric Technic.—The decrease in transmission of light objectively measured by the photoelectric technic agreed well with the clinical estimate in moderate and severe degrees of corneal opacity. The technic was of definite value in the detection of minimal lesions. In several instances, opacities which had been overlooked during life were found to be barely visible to the unaided eye on careful reexamination of the excised cornea after a decrease in transmission was observed by the photoelectric technic. The detection of minimal lesions probably explains the variation in interpretation of results in the preliminary qualitative and in the additional quantitative experiments. Subclinical

lesions, however, would be of little clinical importance, and probably would result in some loss of color vision only, as discussed later.

The photoelectric technic appears to measure the amount of light transmitted; nontransmitted light includes that scattered, which varies with the wavelength of the filter used, as well as that absorbed.[2] The opacities, which appear to act as neutral absorbing filters at all wavelengths, were not homogeneous and were not uniformly distributed over the area of cornea involved. The colorimeter is designed for the measurement of transmission through a homogeneous solution; when a filter is used, measurements are made at a single wavelength. The photoelectric technic as used is not ideal for such experiments, since a method which measures transmission cannot be applied to patients or to intact animals. We have not been able to devise a better method, however.

The use of types of therapy other than are evaluated here are discussed elsewhere.[1]

SUMMARY

Roentgen therapy appeared to be of value in the treatment of acute lesions of ocular vaccinia in experimental animals. The clinical impression that roentgen therapy was of value in decreasing residual corneal opacities was not borne out by measurements of the transmission of light with a photoelectric technic.

Partial immunity of the eye, resulting from previous inoculation of the skin, appeared on the basis of simple observation of experimental animals to decrease the residual corneal opacity of ocular vaccinia. This impression was not confirmed by a statistical comparison of the decrease in transmission of light through the excised cornea of nonimmune and partially immune rabbits, as measured by a photoelectric technic.

The discrepancy between simple observation and statistical comparison of the objective measurements may be due to the detection of minimal or subclinical lesions by the photoelectric technic and to the range in individual variation of the experimental animals.

Bowman Gray School of Medicine.

DISTANCE DISCRIMINATION

III. Effect of Rod Width on Threshold

MONROE J. HIRSCH, Ph.D.
COLUMBUS, OHIO

MILTON W. HOROWITZ, Ph.D.
LAWRENCE, KAN.
AND
FRANK W. WEYMOUTH, Ph.D.
STANFORD UNIVERSITY, CALIF.

THE USUAL form of the rod test for distance discrimination has been designed to exclude all cues of distance except retinal disparity. In spite of this intention, the size cue has not been eliminated. In the Howard test, widely used in the armed forces and in industry, the size cue acts in harmony with, and supplements, retinal disparity. In other tests, of which the "telebinocular" may be taken as an example, size becomes not a secondary, but a confusion, factor. With the "telebinocular" judgments based on retinal disparity are scored as correct responses, while size of the objects enters only to confound the observer. In a normal situation an object looks larger as it approaches the eye; in the "telebinocular" the smallest letter may be, on the basis of binocular parallax, the nearest. This pitting of the size cue against the disparity cue is also a feature of the Verhoeff "stereoptor," in which the subject must judge the relative distances of three objects of different sizes. The intent in these tests obviously is to eliminate size as a basis of judgment. Instructions to disregard size, equally obviously, cannot prevent size from influencing judgment. For these, and other, reasons it is desirable to know the effect of size on binocular distance discrimination.

EXPERIMENT 1

Methods.—The general features of the apparatus have been described in a previous paper.[1] The subject, seated at a distance of 10 meters from the position of instrumental equality, observed the two rods through an aperture 17.5 by 32.5

From the Vision Laboratory, Department of Physiology, Stanford University School of Medicine.

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1. Weymouth, F. W., and Hirsch, M. J.: The Reliability of Certain Tests for Determining Distance Discrimination, Am. J. Psychol. **58**:379-390, 1945.

cm. The rod on his left was stationary and served as a standard of distance. The rod on the right was movable toward or away from the subject by a motor which he started or stopped with a switch; the direction of movement was controlled by the experimenter. Four test conditions were used, in each of which the right, or movable, rod had a diameter of 2 cm.; the left, or standard, rod measured 1, 2, 3 and 4 cm. in conditions *A*, *B*, *C* and *D*, respectively. For all four conditions the inner edges of the rods were separated by 5 cm.

The subjects were 7 university students. Each subject made observations on three days; the first set of observations was considered practice; the second and third sets were recorded and constitute the data presented here. There were rest periods, and no day's observations covered more than one hour. In all, 30 observations were made for each of the four conditions—15 observations for each condition on the second day and 15 observations on the third day. The order of presentation of the conditions was not the same for all observers, nor was it the same for any subject on different days.

The subject looked away from the aperture between observation while the experimenter set the movable rod approximately 300 mm. nearer or farther than the standard. This distance proved in all cases to be above the threshold. At a signal from the experimenter, the subject looked at the rods, answered "near" or "far" and threw the switch setting the rod in motion. The movable rod approached the standard; and when the rods appeared equidistant, the subject stopped the motor and looked away. The direction and distance, expressed in millimeters, of the movable rod from the standard were recorded, and the procedure was repeated.

Results.—The conditions of the experiment gave groups of 15 observations for each subject, two directions of movement and four widths of standard rod, making a total of 840 observations. Each group of estimates of equal distance obtained with this form of the average error method gives a distribution which may be characterized by two statistical measures, the mean and the standard deviation. The mean represents the best estimate of the subject as to the position of equality of the movable and the comparison rod, or position of subjective equality (E_s); the standard deviation (S_x) represents the variability of the settings and has been adopted as the threshold, following Boring. (The psychophysics of distance discrimination is discussed in the first paper of this series.[2]) This threshold is equivalent to the distance from the mean (E_s) to a point cutting off the extreme 16 per cent of the measures.

The mean and the standard deviation for all these groups were calculated. Differences due to the direction of approach proved not to be statistically significant, and the two sets were combined. The means, or positions of subjective equality, are presented in table 1; the standard deviations, or thresholds, in table 2. Considered as standard deviations, the latter are homogeneous, since by Bartlett's test a P of 0.125 was obtained. Treated as thresholds by analysis of variance, they showed no

2. Hirsch, M. J., and Weymouth, F. W.: Distance Discrimination: I. Theoretic Considerations, Arch. Ophth. **39**:210 (Feb.) 1948.

significant differences. On the basis of the average threshold, the angle of binocular disparity amounted to 7.72 seconds. The means, on the other hand, showed significant differences for observers, conditions and interaction when compared with the intraobservation variance. When the more rigorous criterion of the interaction of observer and conditions was used, the variance associated with conditions gave a significant ratio ($P = 0.01$). Thus, it is clear that in spite of the differences in reaction of subjects to the different widths of rods, the conditions exert a significant effect on the position of subjective equality.

TABLE 1.—*Position of Subjective Equality for Each Condition*

Condition	*A*	*B*	*C*	*D*
Left rod size, cm.	1	2	3	4
Right rod size, cm.	2	2	2	2
Subject	Position of Subjective Equality (E_s), Mm.			
A.	− 3.03	−45.77	−66.57	−27.73
B.	−47.43	+ 1.63	−21.40	−45.40
C.	−13.93	−29.93	−29.67	−31.93
G.	+46.07	+43.63	+16.30	+ 7.57
L.	− 8.03	−22.13	−32.83	−40.23
M.	+10.10	+40.83	−42.27	−77.63
O.	+43.50	+ 9.97	−22.97	−26.77
Average	+ 3.89	− 0.25	−28.49	−34.59

TABLE 2.—*Standard Deviation for Each Condition*

Condition	*A*	*B*	*C*	*D*
Left rod size, cm.	1	2	3	4
Right rod size, cm.	2	2	2	2
Subject	Position of Subjective Equality (E_s), Mm.			
A.	68.13	73.33	76.54	86.81
B.	102.32	67.86	70.18	119.93
C.	33.89	39.28	47.18	44.69
G.	75.13	52.21	62.30	45.31
L.	31.21	44.71	33.99	33.37
M.	57.56	61.55	45.86	47.24
O.	47.52	52.71	48.30	42.60
Average	59.39	55.95	54.91	59.99

The values for E_s differed significantly. It will be noticed that the 2 cm. right (movable) rod was set farther than instrumental zero when the fixed rod was smaller, but was set progressively nearer as the width of the fixed rod increased. The data give a linear regression which is significantly different from zero, but for four points it is not safe to try to fix the exact form of the function; all that can be said is that the threshold is a significantly increasing function of rod width. This is what might have been expected; the right rod, when smaller, is placed nearer the observer, a position tending to equalize the visual angles of the two rods.

EXPERIMENT 2

In the preceding experiment the effect of size on the position of subjective equality and the standard deviation (E_s and S_x) was determined. The statistical technic involved was an analysis of variance. The present experiment differed only slightly and affords the possibility of checking the effect of size cue on E_s for single observers.

Methods.—Through an opening 10 by 23 cm. in the partition, two rods were seen against a background of opal glass, transilluminated at a level of 25 millilamberts. The subject, seated 12.7 meters from instrumental zero (the passing point of the rods) and 8 meters from the partition, controlled the motion of the motor-driven movable rod with a switch. At instrumental zero the rods were separated laterally by a distance of 6 cm., corresponding to an angle of 16.3 minutes. The diameters of the rods for the three conditions were:

(*a*) Both rods, 1 cm. (visual angle, 2.7')

(*b*) Right rod, 2 cm. (visual angle, 5.4')
Left rod, 1 cm. (visual angle, 2.7')

(*c*) Right rod, 1 cm. (visual angle, 2.7')
Left rod, 2 cm. (visual angle, 5.4')

The position of the right (movable) rod at the beginning of each trial was approximately 20 cm. from instrumental zero, behind the comparison rod for half the trials and in front of it for the other half. For each of the three conditions, 30 trials were run after a short series of practice trials. The deviation from instrumental zero for each trial was recorded, as plus (+) if the movable rod was set behind the stationary rod and as minus (—) if it was set in front. The 30 trials for each of the three conditions were presented in groups of five, so that any practice effect which had not been eliminated by the practice series would be divided among the three conditions. Five students, 4 sophomores and 1 graduate student, served as observers.

Results.—The mean (position of subjective equality) and the standard deviation (threshold) were calculated for each observer and

TABLE 3.—*Mean (E_s) and Standard Deviation (S_x) for Five Observers for Three Conditions*

	Position of Subjective Equality (E_s), Mm.			Threshold (S_x)		
Observer	Rods of Equal Size	Right Rod Larger	Left Rod Larger	Rods of Equal Size	Right Rod Larger	Left Rod Larger
1.....	+19.233	+19.033	+ 1.433	33.843	32.372	25.969
2.....	+ 1.567	+72.933	—25.000	65.976	50.955	67.782
3.....	— 3.475	+12.100	—22.925	50.638	48.704	66.017
4.....	+ 4.400	+22.700	—29.767	25.844	23.851	31.788
5.....	+ 6.700	+42.200	— 9.000	33.799	35.426	42.920
Average.....	+ 5.685	+33.793	—17.052	42.020	38.262	46.896

for each condition; these data are presented in table 3. When the analysis of variance was applied, results similar to those of the first experiment were obtained. The threshold showed no significant differences in the conditions ($P = 0.20$); the positions of subjective

equality, in contrast, were significantly different ($P = 0.001$). In addition, the differences in E_s for individual subjects were tested for significance; as may be seen from table 4, subject 1 showed a P of 0.02—values for all others were less than 0.01.

The displacement of the E_s was in all cases in harmony with the results of the first experiment; the movable rod, if smaller, was set closer to the observer; if larger, farther, there being in each trial a tendency to equalize the visual angles of the two rods.

COMMENT

The two experiments, although carried out at different times with different observers, gave results agreeing in all essential features. A relatively simple sensory situation was presented, in which distance perception must rest chiefly or exclusively on two clues, retinal disparity and image size. The task is to set a movable rod at the same distance

TABLE 4.—*Differences Between Positions of Subjective Equality (Mean) for Right Rod Larger and for Left Rod Larger*

Subject.	Difference in (E_s), Mm.	P
1	17.600	0.0203
2	97.933	<0.0001
3	85.025	0.0069
4	52.467	<0.0001
5	51.200	<0.0001
Average	50.845	

as the standard rod, and the significant variable is the relative sizes of the rods. The measures are the mean of the settings, or position of subjective equality, E_s, and the standard deviation of the distribution, which is here used as the threshold.

It was found that the threshold shows no significant over-all variability with changes in the size of rods. Although, in the first experiment, with four widths, the situations with the greatest difference in rod size showed larger thresholds, these reached only the 2 per cent level, and could merely be considered suggestive.

In contrast, the position of subjective equality varied significantly with the width of the rod. When the stationary reference rod was wider than the movable rod, the latter was stopped nearer the observer, a sort of "constancy" response in which an attempt is made to equalize the visual angles of the rods in spite of the known difference in size. Although the displacement of E_s increased with the difference in the size of the rods, giving a significant regression, the form of the regression cannot be determined from so small a series of widths.

Although significant, and in the direction reasonably to be expected, these displacements are extremely small. The terms in which they

should be presented and the perceptual mechanism leading to such minute displacements require consideration. Table 5 is an attempt to present the data on E_s in a readily understood form. The first three columns identify the conditions and give the widths of the rods. Column 4 gives the positions of subjective equality. Column 5 gives the position of subjective equality (E_s) referred to the position of subjective equality with rods of equal width. Column 6 gives the value of column 5 as a per cent of the displacement necessary to make the angular sizes of the rods the same; column 7 gives the value of column 5 as a per cent of the average threshold for that experiment. Column 8 gives the difference in angular size resulting from a displacement of the movable rod equal to the value of column 5.

It will be seen that in terms of the displacement required to give equality of angular size, the E_s is negligible, not reaching 1 per cent.

TABLE 5.—*Analysis of Changes in the Position of Subjective Equality (E_s)*

1	2	3	4	5	6	7	8
	Width Fixed Rod, Cm.	Width Movable Rod, Cm.	E_s, Cm.	$E_s - E_=$, Cm.	$\frac{100\ E_s}{D_o}$ %	$\frac{100\ E_s}{Th}$ %	Size Difference, Sec.
A	1	2	+0.389	+0.415	+0.04	+ 7.2	+0.34
B	2	2	−0.025	0.000	.....	.....	.....
C	3	2	−2.849	−2.823	−0.65	−49.0	−1.16
D	4	2	−3.459	−3.434	−0.47	−59.7	−1.42
a	1	1	+0.568	0.000	.....	.....	.....
b	1	2	+3.379	+2.811	+0.22	+66.3	+0.72
c	2	1	−1.706	−2.274	−0.36	−53.6	−0.29

Column 5: Difference between the E_s obtained for the condition and the E_s obtained with rods of equal size ($E_=$).
Column 6: Value of column 5 as a per cent of the total distance required to give equal angular size to the rods.
Column 7: Value of column 5 as a per cent of the average threshold for corresponding condition.
Column 8: Change in angular size of the rod (expressed in seconds of arc) when displaced by the value of column 5.

The difference in angular size corresponding to E_s is, likewise, surprisingly small, in no case equaling 2 seconds, although the corresponding retinal disparities are 7.7 and 3.5 seconds. Perhaps the most significant comparison is with the average threshold for the same experiment. Here, the values range from 7 to 66 per cent.

Although the sensory situation appears to be relatively simple, namely, a pattern consisting of two cues—retinal disparity and angular size in congruent and incongruent relations—the perceptual analysis is not easy. The response is clearly dominated by retinal disparity. The size cue, in the situations in which the rods differ in size, and therefore in which there is an apparent incongruence between the two cues, appears to produce a displacement tending to equalize the visual angles

of the rods. This seems little more than a "token" displacement; but the direction is in all cases consistent, and the greater the difference in size of the rods, the greater the displacement. The maximum displacement is 66 per cent of the threshold, and it will be remembered that the threshold is the standard deviation of the distribution of equality settings. Measured from the mean of this distribution, that is, from the position of subjective equality with rods of equal width, this displacement, or deviation, would include nearly one quarter of the cases. Pitted against the stronger cue of retinal disparity, the size cue may shift the E_s within the original distribution, but seems incapable of displacing the point into a region contradicting the reports of retinal disparity. Of course, it will be obvious that before equality of angular size is actually realized, retinal disparity will have indicated not only "near" or "far" but "double," and the size cue seems unable to override disparity.

There are still practical consequences to be considered. The introduction of the size cue incongruent with the parallax, or disparity, cue has a significant effect on the position of subjective equality but does not significantly affect the threshold. As pointed out in an earlier paper of this series,[2] the E_s is a useful measure of aniseikonia. Experiments with size lenses which support this view will be presented in a subsequent paper.[3] For the present, however, it is sufficient to point out that any testing instrument or method which includes the size cue in a manner incongruent with the parallax cue cannot yield a meaningful E_s, and since this parameter is a measure of aniseikonia, such a method cannot yield any information about aniseikonia. While such instruments as the "telebinocular" and the "stereoptor" give thresholds, it is difficult to state just what is being measured.

In the Howard test the size cue is present, but in a normal, or congruent, fashion. Experiments in this laboratory have shown that under these conditions the resulting position of subjective equality and the threshold are normal. Thus, if both these measures are calculated, information on aniseikonia is obtained, a result not possible with the two tests mentioned.

SUMMARY

With apparatus similar to the Howard test but with rods of different widths, the effect of incongruence between binocular parallax and size on distance discrimination was studied.

The threshold or variability of settings of equality was not significantly affected.

3. Hirsch, M. J.; Horowitz, M. W.; Walker, E. L., and Weymouth, F. W.: Distance Discrimination: IV. Effect of Aniseikonic Lenses, Arch. Ophth., this issue, p. 333.

The position of subjective equality was significantly affected, the position of subjective equality (E_s) being shifted in the direction of size equality to an increasing degree by increasing the difference of width.

The possible perceptual interpretation of this displacement is discussed.

In other papers of this series it has been pointed out that the position of subjective equality is of value as a measure of aniseikonia. Therefore, any test that introduces size as a confusion factor with the intention of "eliminating size" distorts the results ascribed to binocular parallax, renders the test inaccurate and makes it impossible to obtain information on aniseikonia.

Vision Laboratory, Stanford University School of Medicine.

DISTANCE DISCRIMINATION

IV. Effect of Aniseikonic Lenses on Distance Discrimination

MONROE J. HIRSCH, Ph.D.
COLUMBUS, OHIO

MILTON W. HOROWITZ, Ph.D.
LAWRENCE, KAN.

EDWARD L. WALKER, Ph.D.
ANN ARBOR, MICH.
AND
FRANK W. WEYMOUTH, Ph.D.
STANFORD UNIVERSITY, CALIF.

IN THE first paper of this series on distance discrimination [1] it was shown that the results obtained in a test of depth perception might be made to yield two important types of information. If a series of judgments are made, the mean of the distribution expressed in physical units (customarily millimeters) is the position of subjective equality (E_s), and from it may be determined the location of pairs of functionally corresponding retinal points. The standard deviation of the distribution (S_x) may be used to determine the size and relative sensitivity of the pair of corresponding areas. It was pointed out in the paper just mentioned that tests (such as the Howard) which are used today to test distance discrimination are scored in a manner which yields a single measure rather than the two parameters, E_s and S_x. This single measure, while enabling the comparison of members of a group of observers to be made, entails the loss of considerable information. In the third paper of this series,[2] it was pointed out that such tests as the "telebinocular" or the Verhoeff "stereoptor" method not only yield a single measure but, unlike the Howard, cannot be used to give two measures even if the experimenter wished to do so.

The aim of the present experiment was to determine the effect of size lenses on the position of subjective equality (E_s) and the stand-

From the Vision Laboratory, Department of Physiology, Stanford University School of Medicine.

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1. Hirsch, M. J., and Weymouth, F. W.: Distance Discrimination: I. Theoretic Considerations, Arch. Ophth., to be published.

2. Hirsch, M. J.; Horowitz, M. W., and Weymouth, F. W.: Distance Discrimination: III. Effect of Rod Width on Threshold, Arch. Ophth., this issue, p. 325.

ard deviation of the distribution (S_x). Three lenses representing 1, 2 and 3 per cent over-all magnification were used.[3]

METHODS

The variable rod apparatus, formerly described,[4] was used with the angular size of the movable rod constant, thereby eliminating the size cue. The rods were observed at 10 meters, at which distance they were 2.5 cm. wide and were separated by 10 cm. The central portions of the rods were seen through an aperture as a black silhouette against a transilluminated milk glass screen. The experimenter set the right (movable) rod initially at a position noticeably near or far; the subject, who controlled its motion by a switch, set the movable rod equidistant with the standard. The subject was not aware of which lens he was wearing, since the experimenter made the changes in the lens; nor did he know before which eye the size lens was placed, since a plano lens was placed before the eye not having the magnification lens.

Seven conditions of observation were given with the use of a lens of 1, 2 or 3 per cent magnification before either eye combined with a plano lens before

TABLE 1.—*Positions of Subjective Equality (E_s) for Four Subjects for Seven Different Combinations of Size Lenses**

Subject	R_3	R_2	R_1	0	L_1	L_2	L_3
1	—415.98	—273.72	—192.12	— 68.19	— 16.17	+ 50.59	+161.40
2	—119.79	—107.79	— 72.98	+ 31.00	+ 99.83	+200.87	+173.44
3	—368.81	—284.81	—223.83	—128.48	— 16.96	+ 10.85	+ 17.98
4	—178.21	—102.54	— 33.02	+ 30.17	+126.50	+185.94	+262.15
Average	—270.70	—192.22	—130.49	— 33.88	+ 48.30	+112.09	+153.74

* The values of position of subjective equality are expressed in millimeters.

the fellow eye, and of plano lenses before both eyes. The conditions are indicated by the symbols R_3, R_2, R_1, O, L_1, L_2 and L_3. The symbols R and L indicate the eye before which the size lens was placed; the subscript, the per cent of magnification. For each of these seven conditions there were 48 judgments of equality. The mean and the standard deviation (hereafter called threshold) for each of the sets were computed. In all, measurements were made on 4 trained observers. The thresholds are expressed in millimeters, the negative sign indicating that the movable (right) rod was set nearer, and the positive sign, that it was set farther, than the standard.

RESULTS AND COMMENT

The positions of subjective equality and the thresholds, expressed in millimeters, for each observer and for each of the seven conditions, together with averages, are shown in tables 1 and 2. The standard deviations were combined in two different ways; the average threshold

3. The Vision Laboratory received from Prof. Kenneth B. Stoddard, University of California School of Optometry, the three aniseikonic size lenses used in this experiment.

4. Weymouth, F. W., and Hirsch, M. J.: The Reliability of Certain Tests for Determining Distance Discrimination, Am. J. Psychol. **58**:379-390, 1945.

is the arithmetical mean of the four standard deviations, while the combined threshold is the standard deviation for the 192 observations (48 observations made by each of the 4 subjects).

The effect of the lenses on the position of subjective equality was, in general, what might be expected. The magnitude of the E_s which should have resulted from each of the lenses can be calculated. A lens of 3 per cent magnification should displace the E_s 291 mm. at 10 meters; a lens of 2 per cent magnification, 195 mm., and a lens of 1 per cent magnification, 99 mm. For the 4 observers, whose average E_s with no lens was —33.88 mm., resetting the origin to this point yields expected values for the E_s for each of the lenses as follows:

R_3	R_2	R_1	O	L_1	L_2	L_3
—324.88	—229.88	—132.88	—33.88	+65.12	+162.12	+257.12

These calculated values for the E_s and the observed average for 4 observers are presented in figure 1. It will be seen that in each case

TABLE 2.—*Thresholds (S_x) for Four Subjects for Seven Different Combinations of Size Lenses**

Subject	R_3	R_2	R_1	0	L_1	L_2	L_3
1.....	65.77	64.13	59.36	62.66	68.90	69.21	91.62
2.....	152.29	98.19	83.51	103.08	87.41	88.93	208.14
3.....	99.83	83.16	57.89	61.13	68.51	93.05	128.04
4.....	79.78	67.48	88.06	58.67	69.99	77.29	123.48
Average.....	99.17	78.24	72.21	71.38	73.70	82.12	137.82
Combined.....	162.57	117.91	108.42	100.21	98.97	117.08	168.81

* Thresholds are expressed in millimeters.

the values for the E_s lie between the position calculated from the effect of the size lenses and zero, the position of instrumental equality (E_i). This trend of E_s can be fitted by a straight line, the slope of which differs significantly from the calculated values. There is a suggestion that the E_s deviates more from the calculated values with the stronger lenses at either end of the figure, but this is not statistically significant.

The reason for the systematic trend of the E_s toward instrumental zero is probably the presence of some factor other than binocular parallax. Burian [5] described the effect of wearing size lenses for a considerable time (eight to fourteen days). He concluded:

> . . . The disparity factors can be largely disregarded when abundant perspective factors are present in a person's surroundings, but . . . they become immediately operative when the perspective factors are scanty . . . Some persons with normal binocular vision are more apt to let the uniocular factors influence the spatial localization derived from disparity clues than others, in whom the disparity clues dominate.

5. Burian, H. M.: Influence of Prolonged Wearing of Meridional Size Lenses on Spatial Localization, Arch. Ophth. **30**:645-666 (Nov.) 1943.

Since the disparity clue can be disregarded or judgments can be influenced by other cues, it is likely that some factor other than parallax was present and that this was responsible for the trend toward the position of instrumental equality (E_1).

The nature of the factor other than disparity is not clear. Monocular thresholds could not be obtained with the present method; the size cue usually encountered in the rod type of apparatus was eliminated by the gradually changing width of the rod, so that angular size was constant; other monocular cues were eliminated, as they are in all rod tests. It seems most likely that the factor was not one of the cues of distance discrimination, but was the more general time error.

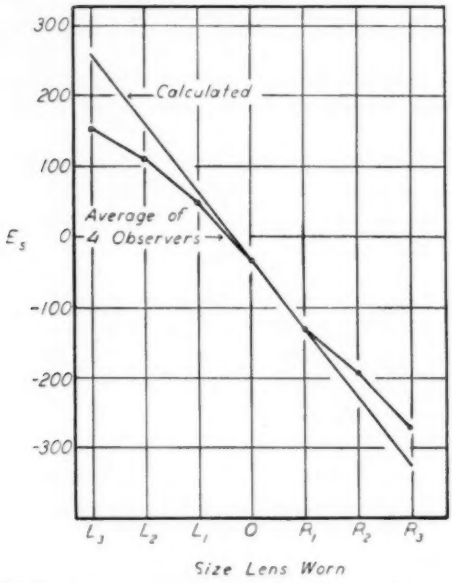


Fig. 1.—Effect of size lenses on the position of subjective equality (E_s).

It was stated earlier that for each judgment the rod was set perceptibly near or far and that the subject's task was to set the rod equidistant with the standard. However, for all lenses the distance was the same. Thus, with the plano lens the time required by the movable rod to reach E_1 was of the order of ten seconds, whether it began at the noticeably near or at the noticeably far position. When the E_s was close to the E_1, the time had no effect. When, however, as in the case of the lens of 3 per cent magnification, the E_s was at some distance from the E_1, approximately five seconds was required to reach instrumental zero from one of the initial positions and fifteen seconds from the other. The tendency would be to overrun in the first case and to underrun in the second, both of which would bring the E_s closer to the E_1.

The average and combined thresholds (S_x) presented in table 2 are shown in figure 2. In general, whether one considers the combined or the average threshold, the standard deviation is lowest for the plano lens or for the 1 per cent lens before either eye and is highest for the 3 per cent lens before either eye. As might be expected, the combined S_x, which includes the variability due to individual differences, is considerably higher for any lens than is the average S_x. The variance ratio of the S_x for either L_3 or R_3 and the S_x for no lens is significantly different to 1 in 1,000. That is, the combined S_x for either of the 3 per cent lenses is significantly higher than the S_X for the plano lens. Analysis of other data accumulated at the Vision Laboratory has yielded a low, but significant, positive correlation between E_s and S_x. The results just described are further evidence of this; the lenses which increase the magnitude of the E_s also increase the magnitude of the S_x.

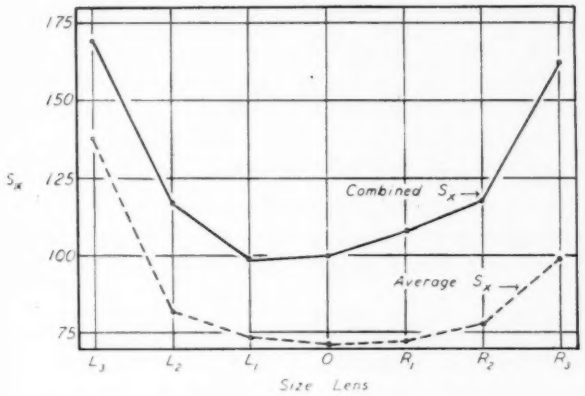


Fig. 2—Effect of size lenses on the average and the combined threshold (S_x).

In the first paper of this series [1] it was stated that the difference between the E_s and the E_1 was a measure of the degree of aniseikonia. In the present experiment, with the use of size lenses, aniseikonia was artificially created and the effect on the E_s was measured. It was found that size lenses alter the E_s in the expected manner, the lower power lenses altering it by the amount that would be expected, and the higher power lenses by an amount less than the calculated. The explanation of this trend toward instrumental zero could be based either on other cues of distance discrimination, which is unlikely, or on the time error, which is probable. The cause need not concern us here, since we are interested primarily in the effect of size lenses on the E_s, and this has been demonstrated to be of the type, if not of the magnitude, which would be expected. It is important that the E_s did vary in a systematic fashion with the use of the size lenses. Therefore,

the rod method of testing depth perception is clearly capable of measuring aniseikonia in the horizontal meridian, provided that the E_s can be calculated. In a former paper [2] it was shown that certain of the tests for distance discrimination, such as the "telebinocular" and the "stereoptor" test, do not yield a position of subjective equality. This is an important consideration in the choice of tests, since the measure (E_s) seems to be a valuable aid in detecting aniseikonia and, with standardization of technic and elimination of the time error, could probably be made to yield a reasonably accurate measure of the degree of aniseikonia.

While a rod type of test would probably not take the place of the eikonometer as a means of determining the degree of aniseikonia, if administered in such a manner as to yield a position of subjective equality (E_s), it would serve as a valuable screening device. It possesses the simplicity and inexpensiveness which are desirable in such tests. The time necessary for the measurement and calculation is relatively short as compared with the many hours necessary to make an eikonometric determination. It should be stressed here that the Howard-Dolman test as used today does not yield this information, since the average deviation from the position of instrumental equality (E_i) is used, rather than the position of subjective equality (E_s) and the threshold (S_x). By increasing the number of observations and by calculating the two measures just mentioned, this test could be made to yield much information about visual function which is at present not made available.

School of Optometry, Ohio State University (Dr. Hirsch).
Department of Psychology, University of Kansas (Dr. Horowitz).
Department of Psychology, University of Michigan (Dr. Walker).
Vision Laboratory, Stanford University School of Medicine (Dr. Weymouth).

RESULTS OF SURGICAL TREATMENT OF CONGENITAL CATARACT

WILLIAM COUNCILMAN OWENS, M.D.
BALTIMORE
AND
WILLIAM F. HUGHES Jr., M.D.
CHICAGO

THERE are few statistical studies on the results of operation for congenital cataract or on the various factors which influence these results.[1] Hence, the best method of treating patients with this defect and the visual result to be expected have not been definitely established.[2] To obtain more information on this subject, a study was made of 148 patients with congenital cataract. The study included an analysis of the results of operations performed on 231 eyes at the Wilmer Ophthalmological Institute between January 1925 and October 1943. The fol-

From the Wilmer Ophthalmological Institute of the Johns Hopkins University and Hospital.

Read before the Section on Ophthalmology at the Ninety-Sixth Annual Session of the American Medical Association, Atlantic City, N. J., June 12, 1947.

1. Horay, G.: Ueber Staroperationen im Kindestalter, Klin. Monatsbl. f. Augenh. **74**:178, 1925. Klare, D.: Klinische Erfahrungen bei der operativen Behandlung des grauen Stares im Kindesalter, Arch. f. Augenh. **102**:165, 1930. Kiss, W.: Sehscharfe der in früher Kindheit doppelseitig Staroperierten, Klin. Monatsbl. f. Augenh. **98**:523, 1937. Falls, H. F.: Developmental Cataracts, Arch. Ophth. **29**:210 (Feb.) 1943.

2. Ziegler, S. H.: Complete V-Shaped Discission for Zonular and Pyramidal Cataracts, J. A. M. A. **77**:1100 (Oct. 1) 1921; Am. J. Ophth. **5**:381, 1922. Bell, G. H.: Operation for Juvenile Cataract in Two Stages, ibid. **9**:433, 1926. Dean, F. W.: Cataracts: Operation for Congenital and Juvenile, Tr. Am. Acad. Ophth. **31**:261, 1926. Alexander, G. F.: The Immediate Removal of Congenital Cataracts, Tr. Ophth. Soc. U. Kingdom **48**:94, 1928. Barkan, O.: A Procedure for Extraction of Congenital, Soft, and Membranous Cataracts, Am. J. Ophth. **15**:117, 1932. Green, J., and Beisbarth, C.: Congenital Cataract Extraction by Method of Barkan, ibid. **16**:603, 1933. Ballantyne, A. J.: Posterior Needling in the Treatment of Lamellar and Other Forms of Soft Cataract, Brit. J. Ophth. **20**: 540, 1936. von Hofe, K.: Beitrag zur Frage der Berufsfähigkeit derjenigen die in der Jugend wegen angeborenen Stars operient wurden, Arch. f. Augenh. **110**: 34, 1937. Flesicher: Zeitpunkt für die operation eines angeboren Stares, Klin. Monatsbl. f. Augenh. **103**:329, 1939. Wolfe, O.: Removal of Soft Cataract by Suction, Arch. Ophth. **26**:127 (July) 1941. Boley, J. P.: Some Practical Points on Congenital Cataract, Bull. Acad. Med. Toronto **18**:197, 1945. Moncreiff, W. F.: Contributions to the Surgery of Congenital Cataract, Am. J. Ophth. **29**:1513, 1946.

low-up period for the group ranged from no return visit to a maximum of twenty-two years, the average period being nineteen months.

The importance of various factors on the final visual result was first studied. These factors were then analyzed for their influence on the occurrence of operative or postoperative complications and on the necessity of secondary operations.

FACTORS INFLUENCING FINAL VISUAL RESULT

Associated Ocular Defects.—The first factor found to be of significance was the presence of associated ocular defects. In this series, 129 eyes (55.8 per cent) had associated ocular defects, and 102 eyes (44.2 per cent) had no associated defects. Nystagmus, monocular strabismus and microphthalmos were the commonest defects (table 1). Monocular strabismus was considered an associated defect only when the operation had been performed on the deviating eye. As a rule, operations

TABLE 1.—*Ocular Defects Associated with 129 Eyes with Congenital Cataract*

Nystagmus alone	53
Monocular strabismus *	27
Microphthalmos	12
Nystagmus and monocular strabismus *	15
Nystagmus and microphthalmos	13
Monocular strabismus and microphthalmos	3
Nystagmus and mononuclear strabismus* and microphthalmos	5
Pigmentary degeneration of retina	1
Total	129

* Operation was always on the squinting eye.

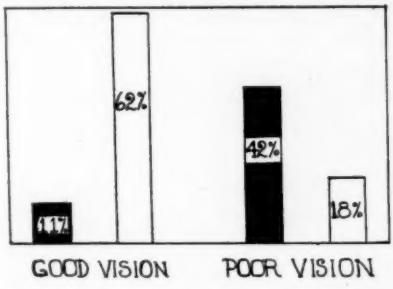
on eyes with strabismus were done on patients with bilateral cataract only after the cataract had been removed from the fixating eye.

The final visual acuity was known in 99 eyes with associated ocular defects and in 93 eyes without associated ocular defects. Good vision, that is, 20/70 or better, was obtained in only 11 per cent of the eyes with associated ocular defects but was obtained in 62.4 per cent of the eyes without such defects (chart). On the other hand, poor vision, a final vision of less than 20/200, resulted in 42.5 per cent of the eyes with ocular defects, but in only 18.3 per cent of the eyes without such defects. Thus, the presence of associated ocular defects was the first, and, indeed, the most important, cause of poor vision.

Age at Operation.—There has been a difference of opinion as to the proper time for the surgical treatment of congenital cataract. Early removal of the cataract has been advocated to prevent amblyopia ex anopsia.[3] On the other hand, postponement of the operation has been recommended because of the greater frequency of postoperative com-

3. Broendstrup, P.: Amblyopia ex Anopsia in Infantile Cataract, Acta ophth. **22**:52, 1944. Goar, E., and Potts, C.: The Relationship of Rubella in the Mother to Congenital Cataracts in the Child, Am. J. Ophth. **29**:566, 1946.

plications thought to accompany early intervention.[4] At first glance, it would appear that this question might be readily answered by a comparison of the results obtained by early operation, i.e., before 2½ years of age, with those obtained by later operation. The answer, however, is not so simple. In evaluating the results obtained with operation at various ages, one must consider the presence of associated ocular defects at these ages. In this series, the eyes operated on at an early age had a higher percentage of ocular defects than those operated on at a later age. Thus, associated ocular defects were present in 72.5 per cent of the eyes in which operation was performed when the patient was 2½ years of age or under, while such defects were present in only 48.6 per cent of the eyes in which operation was deferred until the patient was over 2½ years of age. When the visual results in eyes



Relation of final (postoperative) visual acuity to associated ocular defects in eyes with congenital cataract. Black rectangles show results for eyes with associated ocular defects; plain rectangles, values for eyes without such defects.

having associated ocular defects are separated from those in eyes without defects, the factor of age can be independently evaluated.

The results of such an analysis are shown in table 2. First, of the eyes without associated ocular defects, good vision (20/70 or better) followed operation in only 9.1 per cent when the patient was less than 2½ years of age, whereas good vision was obtained in 69.5 per cent of the eyes when operation was performed after the age of 2½ years. Poor vision (less than 20/200) resulted in 36.3 per cent of the eyes when operation was performed before the age of 2½ years and in only 15.9 per cent when it was done after the age of 2½ years.

4. Wilder, W. H.: The Operative Procedures and Methods of Handling Juvenile Cataracts, Tr. Am. Acad. Ophth., **33**:113, 1928. Knapp, A.: Operative Prognosis of Congenital Cataract, Arch. Ophth. **32**:519 (June) 1944. Moore, F.: Diathermy in Ophthalmology, Tr. Ophth. Soc. U. Kingdom **53**:487, 1933. Shapland, C. D.: Retinal Detachment in Aphakia, ibid. **54**:176, 1934. Philps, S.: Treatment and Post-Operative Care of Cataract in Children, M. Press **216**:376, 1946.

Second, of the eyes with associated ocular defects, good vision resulted in 14.3 per cent of the eyes of patients over 2½ years of age, but in none of the eyes of patients under 2½ years of age. On the other hand, poor vision was obtained in only 35 per cent of the eyes of patients over 2½ years of age and in 68.2 per cent of the eyes of patients less than 2½ years of age at the time of operation. These results indicate that both in patients with and in patients without associated ocular defects, the final visual result is better when operation can be postponed until the patient is at least 2½ years of age.

Type of Cataract.—The maturity of the cataract at the time of operation has been considered an important factor in determining the final visual result. In 52 eyes the cataracts were complete, being entirely

TABLE 2.—*Relation of Age at Operation for Congenital Cataract to Final Vision*

Associated Ocular Defects	Age at Operation, Years	Number with Known Vision	Final Vision		
			20/70+, per Cent	20/100 to 20/200, per Cent	Less than 20/200, per Cent
None	Less than 2½	11	9.1	54.5	36.3
	More than 2½	82	69.5	14.6	15.9
Present	Less than 2½	22	0.0	31.8	68.2
	More than 2½	77	14.3	50.7	35.0

TABLE 3.—*Relation of Type of Cataract to Final Vision*

Type of Cataract	Number with Known Vision	Final Vision		
		20/70+, per Cent	20/100 to 20/200, per Cent	Less than 20/200, per Cent
Complete	45	37.8	22.2	40.0
Incomplete	147	35.4	36.7	27.9

mature in 50 and membranous in 2. In 179 eyes the cataract was incomplete. In all of the latter, portions of the lens were clear. The group of incomplete cataracts included zonular, nuclear, punctate, fusiform, coralliform and anterior and posterior polar cataracts. The series of complete cataracts and the series of incomplete cataracts were entirely comparable in regard to the age of the patient at operation and the presence of associated ocular defects. A direct comparison of the visual results can, therefore, be made. The results of such a comparison are shown in table 3. It was found that the maturity of the cataract had no significant relation to the final visual acuity, the final visual results in the two groups being statistically similar.

Method of Extraction.—In 70 eyes the cataract was removed by one or by repeated simple discissions. The discission was generally made only through the anterior capsule of the lens, but in a few cases

the posterior capsule was also incised. In all cases the lens was stirred after opening the capsule. In 62 eyes the cataract was removed by linear extraction. In these eyes, the anterior chamber was entered with a keratome; a large portion of the anterior capsule of the lens was removed with toothed forceps, and the lens substance was lavaged and expressed from the eye. In 73 eyes the cataract was removed by a combination of discission and subsequent linear extraction. In this group the preliminary discission was followed by a linear extraction after the lens substance and become flocculent, usually three days to two weeks later.

There was no significant relation between the method of operation and the final visual result in the eyes with associated ocular defects

TABLE 4.—*Relation of Operative Method to Final Vision in Eyes with Associated Ocular Defects and in Patients Operated On When Less Than 2½ Years of Age*

Associated Ocular Defects	Method	Number with Known Vision	Final Vision		
			20/70, per Cent	20/100 to 20/200, per Cent	Less than 20/200, per Cent
Eyes With Defects					
Patients less than 2½ years	Discission	11	0.0	27.2	72.8
	Linear extraction	9	0.0	22.2	77.8
	Discission followed by subsequent linear extraction	2	0.0	100.0	0.0
Patients over 2½ years	Discission	20	10.0	50.0	40.0
	Linear extraction	19	10.5	42.0	47.5
	Discission followed by subsequent linear extraction	22	18.2	54.5	27.3
Eyes Without Defects					
Patients less than 2½ years	Discission	4	25.0	25.0	50.0
	Linear extraction	3	0.0	66.0	33.0
	Discission followed by subsequent linear extraction	4	0.0	75.0	25.0

or in the eyes in which operation had been performed while the patient was less than 2½ years of age. The statistically similar visual results in these two groups are shown in table 4.

However, in the eyes without associated ocular defects, when the operation was performed after the patient was 2½ years of age, there was a significant relation between the method of operation and the final vision. These results are shown in table 5. Linear extraction or a combination of discission and subsequent linear extraction was superior, giving good visual results in 76.3 and 78.4 per cent of the eyes, respectively, whereas simple discission or repeated discission alone gave good visual results in only 28.6 per cent.

Intracapsular extraction was attempted in 14 eyes and was successfully accomplished in 8. Of the patients for whom the operation was successful, 1 was 3 years old; 3 were between 20 and 30 years of age,

and 4 were over 30 years of age. Three patients were without associated ocular defects, and all these obtained a final vision of 20/30 or better. Of the 5 patients with successful intracapsular extractions who had associated ocular defects, 2 obtained 20/70 vision; 2, 20/200 vision, and 1, vision of less than 20/200. Of the 6 patients in whom the capsule ruptured, 1 was between 20 and 30 years of age, and 5 were over 30 years of age. Of the 3 of these patients without associated ocular defects, 2 obtained final vision of 20/20 or better, and 1 had a final visual acuity of 20/200. Of the 3 patients with associated ocular defects, 2 obtained 20/100 vision, and 1, 20/200 vision. It appears, therefore, that in this small series rupture of the capsule at operation had no appreciable effect on the final vision.

Extracapsular extraction was performed on 11 eyes. Four eyes had no associated ocular defects, and good vision was obtained in 2 of them.

TABLE 5.—*Relation of Method of Operation to Final Vision in Eyes with No Associated Ocular Defects Operated On When Patient Was Over 2½ Years of Age*

Method of Operation	Number with Known Vision	Final Vision		
		20/70+, per Cent	20/100 to 20/200, per Cent	Less than 20/200, per Cent
Discission	14	28.6	35.7	35.7
Linear extraction	21	76.3	9.5	14.3
Discission followed by subsequent linear extraction	37	78.4	13.5	8.1

COMPLICATIONS

The first section of table 6 shows the incidence of various complications in eyes with associated ocular defects and in eyes without such defects. There was no statistical difference in the incidence of complications in these two groups. Similarly, there was no significant relation between the occurrence of complications and the age of the patient at the time of operation (table 6, second section). Likewise, there was no difference in the incidence of complications in patients with mature and in patients with immature cataract (table 6, third section).

Table 7 shows the relation of operative and postoperative complications to the method of operation. The higher incidence of loss of vitreous with linear extraction and with discission and subsequent linear extraction is, of course, a reflection of the wider opening of the anterior chamber and the manipulations required in these operations. Other complications occurred with almost equal frequency with the three methods of cataract extraction: simple discission, linear extraction and discission with subsequent linear extraction.

Of the 14 cases of attempted intracapsular extraction, loss of vitreous occurred in 4, secondary glaucoma developed in 2 and postoperative iridocyclitis occurred in 1. Thus, in this small series, there was no increase in the incidence of iridocyclitis, but loss of vitreous and secondary glaucoma were definitely more frequent than with the other methods of extraction.

Gaping of the wound and prolapse of the iris were infrequent complications. In only 2 cases in the entire series did gaping of the wound occur, and in only 1 did prolapse of the iris develop.

TABLE 6.—*Incidence of Complications*

	Number of Cases	Loss of Vitreous, per Cent	Hemorrhage in Anterior Chamber, per Cent	Iridocyclitis, per Cent	Endophthalmitis, per Cent	Secondary Glaucoma, per Cent	Detachment of Retina, per Cent	Phthisis Bulbi, per Cent
Associated Ocular Defects								
None	102	16.7	5.9	3.9	2.9	0.0	2.0	9.8
Present	129	10.1	3.1	6.2	0.0	5.4	1.6	3.9
Age at Operation (Years)								
Less than 2½	58	5.2	3.5	3.5	0.0	1.7	1.7	8.6
Over 2½	173	15.6	4.6	5.8	1.7	3.5	1.7	5.8
Type of Cataract								
Complete	52	9.6	3.9	11.5	0.0	1.9	1.9	7.7
Incomplete	179	14.0	4.5	3.4	1.7	3.4	1.7	6.1

TABLE 7.—*Relation of Complications to Method of Operation*

Method of Operation	Number of Cases	Loss of Vitreous, per Cent	Hemorrhage in Anterior Chamber, per Cent	Iridocyclitis, per Cent	Endophthalmitis, per Cent	Secondary Glaucoma, per Cent	Detachment of Retina, per Cent	Phthisis Bulbi, per Cent
Discission	70	1.4	0.0	4.3	1.4	1.4	0.0	7.1
Linear extraction	62	16.1	6.5	8.1	1.6	3.2	3.2	8.1
Discission followed by linear extraction	73	17.8	6.9	0.0	1.4	2.7	2.7	5.5

Purulent endophthalmitis developed in 3 eyes in the series (1.3 per cent). In all cases it occurred before the sulfonamide drugs or antibiotic substances were available and phthisis bulbi developed in all.

Nonpurulent iridocyclitis developed in 12 eyes (5.2 per cent) but subsided in all. Three of the eyes obtained good vision (20/70 or better). In 5 eyes numerous secondary operations were performed to clear the pupillary opening. In these eyes the iridocyclitis recurred, phthisis bulbi finally developing.

To determine the importance of sensitivity to retained lens material as a cause of postoperative nonpurulent iridocyclitis, intracutaneous

tests with lens protein were performed on 8 of the patients. In 3 there was strong cutaneous sensitivity to the protein, while in 5 the tests gave negative results. Nineteen control tests, on patients without postoperative iridocyclitis, were performed. None of these gave positive cutaneous reactions to the injected lens protein. In the 3 patients with nonpurulent iridocyclitis and pronounced cutaneous sensitivity to lens protein, the inflammation was thought to be endophthalmitis phacoanaphylactica. Two of these were desensitized to lens protein [5] before an operation was undertaken on the fellow eye. In each case, after the cutaneous sensitivity had been abolished the operation on the fellow eye was not followed with iridocyclitis.

Detachment of the retina occurred in 4 eyes (1.7 per cent). In 2 eyes operations for retinal detachment were performed. In one of these eyes the operation was successful, the patient obtaining 20/200 vision. In the other, the retina could not be reattached.

TABLE 8.—*Relation of Method of Operation to Subsequent Operations*

Method of Operation	Number of Cases	Subsequent Operations, per Cent			
		Discission	Capsulotomy	Capsulectomy	Total
Discission	70	40.0	15.7	10.0	65.7
Linear extraction	62	17.8	16.1	3.2	37.1
Discission followed by linear extraction	73	23.3	8.2	5.5	37.0

Secondary glaucoma occurred in 7 eyes (3.0 per cent). In 1 eye the glaucoma was transient, the tension being controlled without miotics. In 2 eyes the tension was controlled with miotics alone, while a third required cyclodialysis in addition. In the 3 remaining eyes the ocular tension could not be controlled even with miotics or operations.

SECONDARY OPERATIONS

No significant correlation was found between blocking of the pupil by capsular remains, with the consequent necessity of secondary operations to clear the pupillary space, and any of the following factors: presence or absence of associated ocular defects, age of the patient or maturity of the cataract at the time of the initial operation. However, when the initial operation was a simple discission, repeated discissions to clear the pupillary space were required in 40 per cent of the eyes. Table 8 shows that the frequency of secondary operations to clear the pupillary space was significantly higher (65.7 per cent) in the eyes operated on by discission alone than in the eyes operated on by linear extraction or by discission with subsequent linear traction (37.0 per cent)

5. Hughes, W. F., Jr., and Owens, W. C.: Postoperative Complications of Cataract Extraction, Arch. Ophth. **38**:577 (Nov.) 1947.

COMMENT

It is well known that the final visual result following removal of congenital cataract is much poorer than the result following the extraction of senile cataract. This has been attributed to the higher incidence of operative and postoperative complications thought to follow operations for congenital cataract. Such an increased liability to complications was not found in this series. Table 9 shows the incidence of operative and postoperative complications in the present series of cases of congenital cataract and in the series of cases of operations for senile cataracts previously reported.[6] In these two series, there was no significant difference in the incidence of complications in the operations performed for congenital cataracts and in those performed for senile cataract. Therefore, the poor visual results following the removal of congenital cataract cannot be attributed to more frequent complications.

What other factor can account for the poor vision following the removal of congenital cataract? Gross associated ocular defects were

TABLE 9.—*Relative Incidence of Complications in Operations for Congenital and Senile Cataracts*

Type of Cataract	Number of Cases	Loss of Vitreous, per Cent	Hemorrhage in Anterior Chamber, per Cent	Iridocyclitis, per Cent	Endophthalmitis, per Cent	Secondary Glaucoma, per Cent	Detachment of the Retina, per Cent
Congenital	231	17.3	4.3	5.2	2.2	2.2	1.7
Senile	1,650	8.2	9.1	6.7	1.2	4.1	1.5

present in 55 per cent of the eyes with congenital cataract. In these eyes the final visual result was very poor, vision of 20/70 or better being obtained in only 11 per cent. However, even in the group with no gross associated ocular defects, the final visual results were poorer than those following the removal of senile cataract. In the present series the removal of congenital cataract from eyes with no gross associated ocular defects resulted in good vision (20/70 or better) in only 62.4 per cent. In the series previously reported the removal of senile cataract resulted in good vision in from 80 to 95 per cent of the eyes, according to the operative technics employed. Since the occurrence of complications following the removal of congenital cataract is essentially the same as that following the removal of senile cataract, it is possible that the eyes with congenital cataract and no gross associated ocular defects have obscure defects, such as incomplete or abnormal formation of the retinal elements. It is likely, therefore, that the overall poor visual results following removal of congenital cataracts must

6. Hughes, W. F., Jr., and Owens, W. C.: Extraction of Senile Cataract, Am. J. Ophth. **28**:40, 1945.

be attributed to the fact that many of the eyes with congenital cataract are otherwise defective and cannot be expected to have high visual acuity even when the cataracts are successfully removed.

SUMMARY AND CONCLUSIONS

Gross associated ocular defects were present in 129 of 231 eyes with congenital cataract. The visual results in these eyes were significantly poorer than in the eyes without such gross ocular defects.

The visual results for the patients on whom the operation was performed before 2½ years of age were poorer than for those operated on after the age of 2½ years.

The visual results were essentially the same whether the congenital cataract was complete or incomplete at the time of operation.

Linear extraction or discission with subsequent linear extraction produced better visual results than simple discission in the patients with no associated ocular defects who were operated on after 2½ years of age.

Operative and postoperative complications were as frequent in the eyes without associated ocular defects as in the eyes with associated ocular defects. No significant relation was found between the occurrence of complications and the age of the patient or the maturity of the cataract at the time of operation.

The type of operation performed had no significant relation to the occurrence of postoperative complications.

The number of secondary operations necessary to clear the pupillary space was higher after simple discission than after linear extraction or discission with subsequent linear extraction.

Complications occur with essentially the same frequency after the extraction of senile as after extraction of congenital cataract. The poorer visual results after extraction of congenital cataract are attributed to either gross or obscure associated ocular defects.

Wilmer Ophthalmological Institute (Dr. Owens).

University of Illinois School of Medicine (Dr. Hughes).

ABSTRACT OF DISCUSSION

Dr. Frederick C. Cordes, San Francisco: The authors found associated ocular defects in 55.8 per cent of the eyes with congenital cataract. This corresponds rather closely with the figures given by Fall, who found associated ocular defects in 52.6 per cent of a series of 131 patients. Thus, it is apparent that the prognosis must be guarded in the surgical treatment of congenital cataracts.

A visual acuity of 20/40 or better would seem to be sufficient to contraindicate operation, since the wearing of the heavy, disfiguring lenses is in itself a handicap to the growing child. In some instances,

as with the lamellar cataract, the opacity may increase in density so that operation may be necessary later.

The statistics of Owens and Hughes would seem to indicate that operation should not be performed before the age of 2½ years because of the poorer results. Horay, in reporting the results in 282 operations for congenital cataract, stated that he considered unfounded the opinion that operation should be done after the first year. In his series the results were just as good for operations done in the first year of life as later. The failures during the first year were often the result of other congenital anomalies. Fuchs stated:

> The earlier the operation the better the visual result. . . . Eyes with congenital cataract are usually smaller than normal and, if the lens is removed early, continue to be retarded in their growth. If one waits until later to operate, the eye will have increased some in size, but the visual acuity will be much worse because the retina, owing to lack of use, fails to develop normally.

From personal experience and from the literature, it would seem that in those cases of bilateral cataract in which the opacities are sufficiently dense to interfere with the development of fixation, the first eye should be operated on at the age of 6 months so that the child may develop this faculty, and thus perhaps avoid nystagmus.

Only approximately 50 per cent of these eye have a postoperative vision of 20/70 or better. After the operation the child has to learn to see, as he lacks visual memories. Thus, vision must be developed through experience. In 1927 Fuchs said:

> One reads in romances how the congenitally blinded person is operated on, and how at the first removal of the dressings he is surprised at the beauty or homeliness of his adored ones. This must be regarded purely poetic fantasy.

The authors found that linear extraction or a combination of discission and subsequent linear extraction was superior to simple discission or repeated discissions. This is in agreement with Falls's experience, who in his series found that the best visual acuity was obtained when discission and linear extraction were used. Horay concluded that the operation of choice, irrespective of the age of the child, was a linear extraction done at one sitting. Thus, these three large series of cases indicate that repeated discission gives the poorest results.

My personal choice of operation in cases of soft congenital cataract is the linear extraction as modified by Otto Barkan. The important modifications are as follows: 1. Injection of epinephrine hydrochloride (1:1,000) at the limbus, at the site of the incision, to prevent the iris from becoming involved. It also keeps the pupil widely dilated during the operation. 2. Placing of the incision parallel to the surface of the iris and 1 to 2 mm. inside the cornea so as to obtain a valvelike incision. In addition, it has been found that the injection of air into the anterior chamber at the completion of the operation is an important factor in the prevention of anterior synechias and incarceration of the iris.

With certain types of congenital cataract, particularly the congenital membranous cataract, the discission as advocated by Wheeler is indicated.

In cases of the disk-shaped cataract, in which two types are present—a membranous central portion and a peripheral soft cataract—a discis-

sion followed later by an incision through the membrane with Berens or de Wecker scissors is usually necessary.

In cases of cataract following rubella, in addition to the consideration of other possible anomalies, it is well to remember that the absorption of the lens substance is slower than that of the usual congenital cataract and that in most instances it is difficult to dilate the pupil with any mydriatic.

DR. WILLIAM C. OWENS, Baltimore: It was a great pleasure to have Dr. Cordes discuss this subject, since he has been interested in it for many years. I think that in essence he agrees with our results except for the question of the age at operation. As he stated, this subject has been a controversial one for many years. Further work will be necessary before the final answer will be available.

EFFECT OF A PRISM ON THE CORNEAL LIGHT REFLEX

EMANUEL KRIMSKY, M.D.
BROOKLYN

ONE IS accustomed to think of a prism as a wedge-shaped piece of glass which bends a ray of light traversing it in the direction of its base. That this ray of light affects the position of the corneal light reflex in a paradoxic manner is not generally known.

Displacement of a corneal light reflex from a fixational position is known to indicate ocular deviation, and any artificial method which could restore such displacement to a fixational position can likewise serve to register the amount of such deviation. Such artificial restoration by binocular instruments employing flexibly moving lighted targets through a measured range is established practice. That a corrective prism could likewise restore a displaced corneal light reflex to a fixational position was noted by me in a previous report.[1]

Maddox,[2] in his excellent treatise entitled "The Clinical Use of Prisms," devoted a chapter to the internal reflection of images through a prism without suggesting its possible effect on the corneal light reflex. He rightfully stated [2] that "hitherto we have only considered the refraction of light by prisms, but at each surface a smaller portion is also reflected." He noted:

> . . . the faint image of a flame viewed through the center of a prism points exactly to the apex of the prism, and it is curious that so simple a guide does not appear to have been hitherto utilized. Could we not utilize also the distance between the bright image of a flame and its faint image for measuring the strength of a prism?

Maddox made no mention whatever of the effect of a prism on the corneal light reflex. Although more than a half-century has passed since the publication of this memorable treatise, I take the liberty of repeating Maddox's words by stating that it is curious that so simple a guide as the artificial displacement of a light reflex on a cornea or a convex spherical mirror by a prism does not appear to have yet been utilized or reported in textbooks on optics or ophthalmology.

If one accepts the dictum that artificial restoration of the corneal light reflex from a displaced to a central or fixational position is a

1. Krimsky, E.: The Fixational Corneal Light Reflex as an Aid in Binocular Investigation, Arch. Ophth. **30**:505-521 (Oct.) 1943.

2. Maddox, E.: The Clinical Use of Prisms, London, John Wright & Sons, Ltd., 1893.

prerequisite to the measurement of squint, one must likewise think of a prism as a medium which should also produce such a corrective effect. In a previous report, I emphasized that such displacement must be toward the apex of the prism, and not the base. If, for example, one thinks of esotropia as registering temporal displacement of the corneal light because the eye is turned in, one might make the mistake of assuming that a base-out prism, which bends a ray of light toward its base, would displace the light reflex still more temporally in the direction of the base. If such a phenomenon were possible, one would be at a loss to explain binocular fixation in terms of centralized corneal light reflexes. If such an apparently strange phenomenon of apical displacement of a corneal light reflex does occur with prism, it is because with such a light reflex one thinks in terms not of refraction but of reflection of light or image displacement, analogous to looking through a prism and seeing an image displaced apically.

To simplify matters, one may regard the cornea as a convex spherical mirror or a reflecting medium. As a curved mirror, it is composed of a multitude of plane mirrors or tangents to this curved surface. To make matters still simpler, one may begin with a plane mirror and see how its reflecting properties are modified when it is rotated (deviated) and then when a prism is interposed.

LAWS OF REFLECTION IN RELATION TO A PLANE MIRROR [3]

Not only will a light the rays of which cross or strike a plane mirror yield a virtual, erect image, but this image will appear to be as much behind the mirror as the object light is in front of it. Moreover, this object light will produce an image of exactly the same size.

If the examiner's testing eye (or camera lens) and the light source are in direct line and perpendicular to the axis of mirror (fig. 1), the image will likewise appear in a continuous line with the observer's eye. Though the light that emanates from a flashlight consists of pencils or rays of light which spread out in different directions, it is only the particular wave or waves in direct line with the mirror which govern the image response as seen in the mirror. If a flashlight is tilted at a considerable angle and directed almost parallel to, instead of perpendicular to, the surface of the mirror, it is still those selective rays perpendicular to the surface of the mirror which govern the so-called light reflex in the corneal mirror.

As the light is moved back and forth from the mirror in a direct perpendicular axis, one sees a corresponding fore-and-aft displacement of the light reflex in the mirror. With a corneal convex mirror axial displacement would hardly be apparent because of the very small radius of curvature of the mirror.

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3. Cowan, A.: Ophthalmic Optics, Philadelphia, F. A. Davis Company, 1927.

If the flashlight is kept in direct perpendicular axis to the mirror, but the observer's eye or camera lens is shifted to one side, the light reflex will no longer appear to be coming straight toward him, but will appear to be coming from a side. This parallactic displacement of the image in a plane mirror is greater than that in a spherical mirror, even though the axial relationship of the object light and the image light is not disturbed. The eye of the observer, by shifting its position away from this fixed perpendicular axis, sees the reflex image likewise as displaced.

Displacement of the reflex image in a mirror can also be produced by rotating the mirror on its vertical axis or edge through a central rotating point without upsetting the fixed relation of the camera lens or the testing eye behind the flashlight. Whether one rotates the mirror

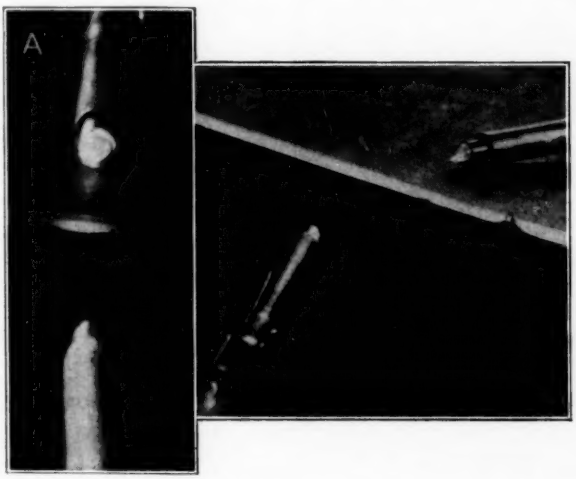


Fig. 1.—Reflection by a plane mirror. (*A*) The light seen through a mirror placed at right angles to the source of light will be seen to register a light reflection, or "reflex," directly opposite, or facing, the object light. The same effect is observed when the examiner sees the light reflex in a fixing eye with his eye directly behind the test light.

(*B*) Without change in the positions of the observer's eye and the test light, the mirror is now tilted on its edge or "rotated." The light reflection is now seen to move to one side, in the same way as though the eye were rotated from its position of fixation. In this case, the light reflex is displaced to the right of the object light in the same way as though the left eye facing the examiner were to rotate to the right.

As a plane or a convex mirror is rotated, one observes the position of the light reflection to change accordingly. The examiner translates this displacement in terms of the new direct path between his eye and the reflection at the principal focus, where it intersects the corneal mirror.

(or merely rotates an eyeball) or/and changes the position of the eye behind the test light, the position of the reflex image is based on the simple law of the angle of incidence and the angle of reflection (figs. 1 and 2).

DISPLACEMENT OF IMAGE BY PRISM VERSUS MIRROR

The effect of rotating a plane mirror on its edge, or vertical axis, in producing displacement of an image is not unlike what one would find in looking through a prism. One can note, furthermore, that such rotation of a mirror through a measured amount will produce displacement of an image equal to that produced by a prism of a strength equivalent to the angle of rotation. For example, if it is assumed that 20 arc degrees equals roughly 37 △, then a mirror rotated around a central vertical axis over a protractor reading of 20 degrees will produce an equivalent image displacement. Rotation of the mirror which

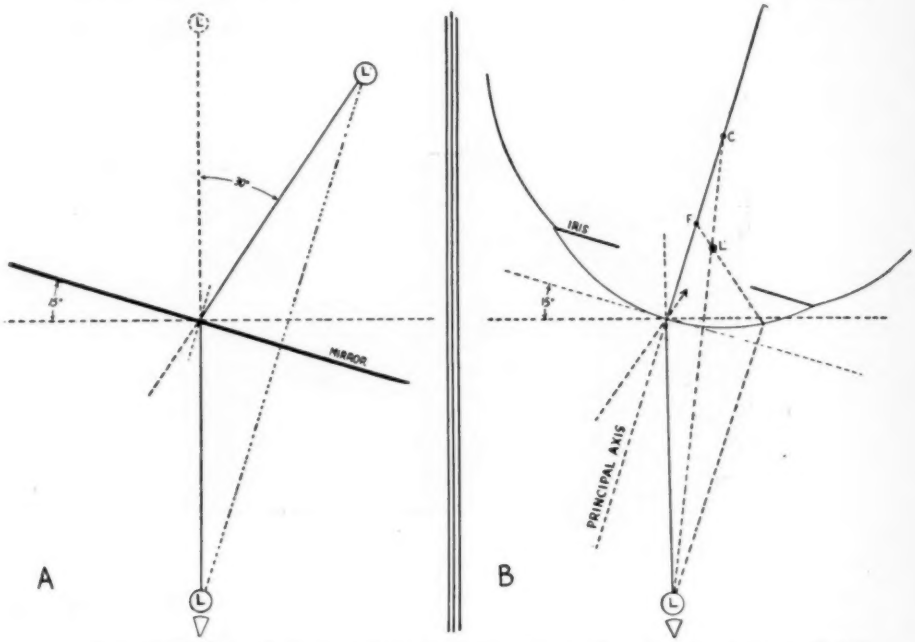


Fig. 2.—Effect of mirror "rotation" on the position of the light reflection *A,* with plane mirror; *B,* with convex mirror (cornea).

brings the right half nearer the camera will also produce reflex displacement to the right (figs. 1 and 2). The apex of the prism placed to the right with the flashlight at right angles to the mirror will produce the same effect. A combination of prism and mirror in such positions would produce a summation of effect. However, placement of prism and mirror in reverse positions will have a neutralizing effect, and not only will the reflex image be directed toward the apex but the direction of the flashlight axis as seen through the prism will appear rectified in a straight continuous line with that of the object light; in other words, the angulation of the lines as a result of mirror rotation will be trans-

formed into a continuous straight line, and the flashlight image will appear to be coming directly toward the observer's eye and will no longer be angulated. This phenomenon (fig. 3) is interesting, for it serves to indicate that the effect of a prism is not merely to restore a reflex image to a central, or fixational, position but also to restore the image of the axis to a continuous straight line. This phenomenon, moreover, is of interest because in actual ocular deviation one learns to think of a prism as a medium not only for restoring the light reflex to a central, or fixational, corneal position but also for rectifying the image along this bent visual axis and making the eyeball actually appear straight.

REFLECTION FROM A CONVEX MIRROR

If one can conceive of a spherical mirror or the eye as a collection of plane mirrors arranged tangentially around different points of the circle, it becomes easier to understand the disposition of the lighted image or of reflections behind its surface. A luminous spot directed toward a convex mirror will, provided the eye of the observer is behind the test light, be seen to proceed through the mirror without interruption to focus at a point halfway between the surface of the sphere and the center of rotation. This point is known as the principal focus. Why does the light reflex take its position at this point? It is because the divergent rays reflected from any tangent on the convex sphere will be reflected back not to the center of the sphere but to the aforementioned midway point, which is called the principal focus. Unlike the image in the plane mirror, the image as seen through a convex spherical mirror is also virtual and erect but is nearer the surface than the object light. The smaller the radius of curvature, the closer to the surface this light reflex becomes, although as long as the surface is a sphere the light reflex is at or near the principal focus, or halfway between the center of curvature and its surface.

As in the case of the plane mirror one would like to know how the light reflex image is displaced when the testing eye alone is moved away from the principal axis along which the light stimulus is directed. Then one might wish to know the variations in position of the light reflex when the testing eye alone is in line with the principal axis and only the test light is moved into different positions. In the latter instance, with the testing eye in line with the principal axis, the respective positions of the light reflex as a result of such changing positions of the test light are shown in figure 7. Only when the light is in a direct line with that of observer and patient will it after reflection return along the same path. Light moved to any other point will be seen to move in a corresponding direction, although through a smaller linear range than in the case of the plane mirror.

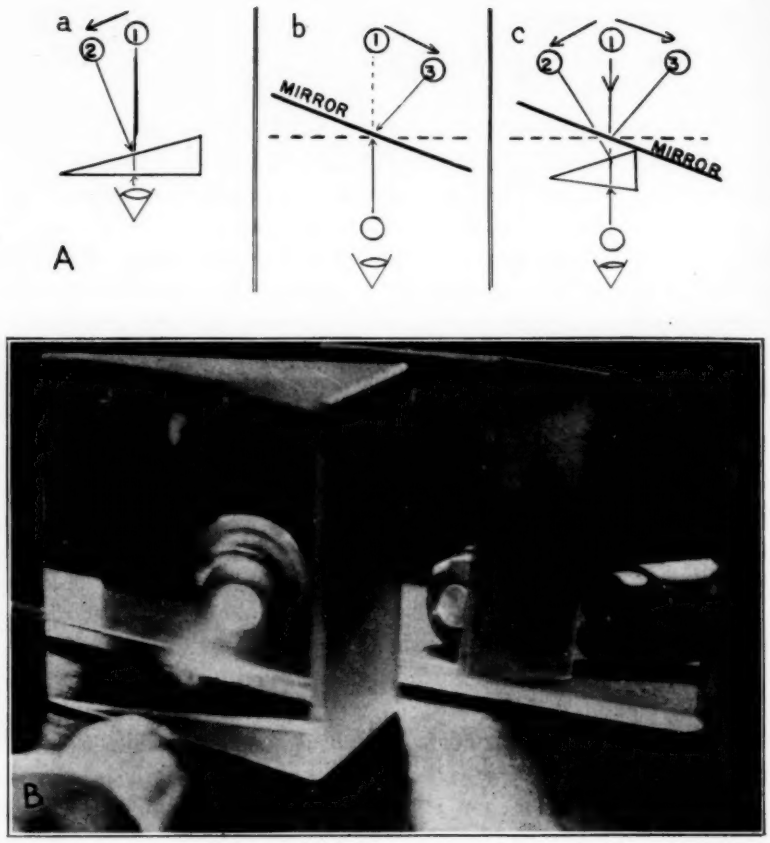


Fig. 3.—Displacement of image or light reflection by prism as compared with displacement by mirror. *A*, schematic drawings; *B*, photographic demonstration.

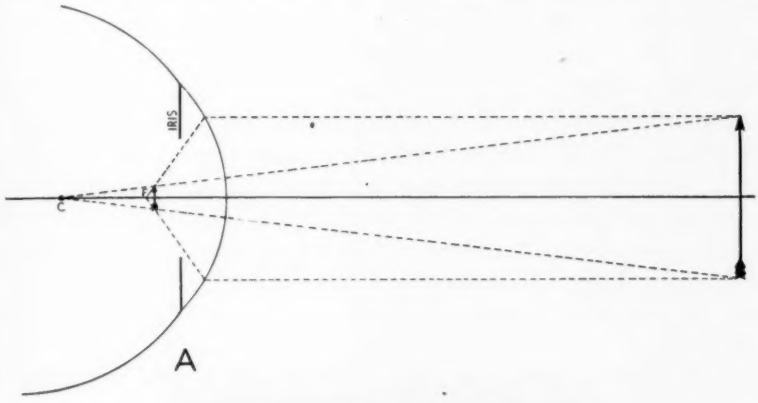
(*A*) In *a*, the image as seen through the prism shifts from position *1* obliquely forward and to the left, or toward the apex of the prism to position *2*. In *b*, a mirror is substituted for a prism and is so "rotated" on its edge (as in fig. 1 *B*) as to shift the light reflection obliquely forward in the opposite direction. In *c*, the effect of both mirror and prism placed in the aforementioned positions serves to restore the light "reflex" to a central position by prism and mirror counteracting the effects of each other.

(*B*) This photograph portrays vividly the mechanism of the prism reflex test. The object test light is in the lower left corner. The plane mirror is tilted at an angle, as in figure 1 *B*, with the right half nearer the camera. Therefore, when one ignores the prism, the light reflection by mirror alone is the farthest to the right. That reflection is comparable to the reflex in the left eye as seen by the observer to be rotated to his right, thereby causing apparent displacement of the light reflex temporally. The plane mirror is comparable to the cornea.

A square prism is placed in front of this mirror, and an image of this prism is also observed in the mirror. One also notes three light "reflexes," the aforementioned one, without the prism; a second, seen through the image prism alone, and a third, seen through the object prism. Observe that the light reflex is displaced toward the apex of the prism, rather than toward the base. Note also that the third "reflex," through the object prism, appears to be coming face to face with the object light. This phenomenon is especially interesting and significant, for it shows that the action of a prism is not merely to shift the image in the direction of the apex of the prism, but also to rectify the course of this image, or to neutralize the tilting of the mirror. The three reflexes are interdependent. Which of the three reflexes determines the deviating property of the prism? The answer is: the third, or object "reflex." The second, or middle, "reflex" displaces the image only halfway through the image prism. The third, or object prism, "reflex" represents the added displacement of the image prism through the object prism. Note the edge of the image prism through the object prism. In clinical testing with prisms one always looks through the object prism instinctively to observe the light reflex.

POSITION OF THE CORNEAL LIGHT REFLEX

If one is to think of the cornea as a convex spherical mirror, light rays which strike it should meet at or near a common point. This point is the principal focus of the cornea and is situated about 4 mm. behind its



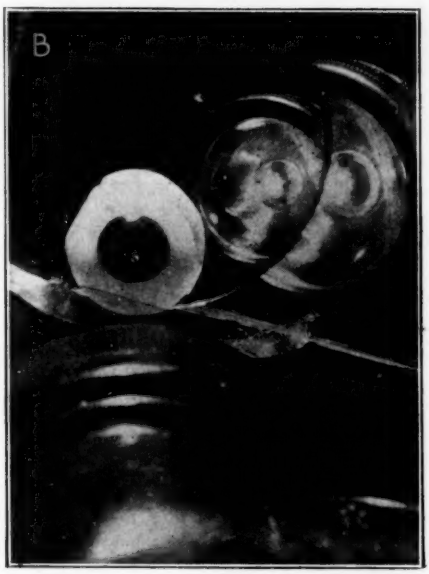


Fig. 4.—Relative sizes and positions of images or light reflections with convex and plane mirrors, respectively.

A, diagram illustrating that the corneal light reflex is in reality a subcorneal light reflection of reduced size, and showing that a prism placed in front of an eye registers a reduced size of image prism behind the cornea.

B, photograph showing comparative sizes of light reflections in plane and convex mirrors, respectively. In the plane mirror, the image size is the same as the object size; in the convex mirror over it, the image of the flashlight is smaller, and, finally, in the corneal convex mirror, of very short radius, the light reflex is rather small.

anterior surface, it being assumed that the cornea has a radius of curvature of 8 mm. Hence, the term corneal light reflex is a misnomer because the light reflex is subcorneal (fig. 4) in position and technically is a reflection rather than a reflex.

A light that strikes the eyeball at an angle is seen to produce several images, known as the images of Purkinje because of structural components, such as the lens, seen in the path of the light rays.[4] The familiar corneal light reflex is therefore technically but one of these five or six Purkinje images and is said to be deposited in the region of the anterior surface of the lens. It is the brightest of these images and is erect and virtual. Because it also exists in an aphakic eye, I prefer to discount the corneal light reflex in terms of Purkinje images and to think of it solely in terms of a principal focus image, like that which one might find with any spherical ball. Its relation to the anterior surface of the lens is merely incidental, because that is where one finds the principal focus of the cornea.

The intersection of the different points from parallel rays will determine both the position and the size of the image. The importance of appreciating the position of the light reflex in relation to the position of the observer's eye may be illustrated by the following mathematical example:

An eye fixing a point source of light at a distance of 33 cm. is found to register a corneal light reflex on the center of the pupil as long as the observer's eye is behind the test light. Where would the light reflex appear to be on the cornea if the observer's eye were moved 60 degrees along the arc of the anglometer while the examinee's eye maintains fixation on the light source? In figure 5 the light reflection is behind the cornea at a point on the principal axis halfway between the cornea and the center of curvature. When an eye is moved 60 degrees along the arc, the corneal light reflection will appear to move from point S to point O on the cornea. The following formulas with the aid of the diagram (fig. 5) are self explanatory.

Angle $OFS = 60°$ $\qquad \sin B = \frac{b \sin A}{a}$

$SF = 4$ mm. $\qquad \sin B = \frac{4 \text{ (mm.)} \times 0.866 \text{ (sin } A\text{)}}{8 \text{ (mm.)}}$

$FC = 4$ mm. $\qquad \sin B = 0.433 = 25° 42'$

$OC = 8$ mm.

Angle $OFC = 120°$ $\qquad$ Angle $C = 180° - (120° + 25° 42') = 34° 18'$

which is equivalent to 4.7 mm. off center of the cornea, based on a corneal circumference of about 50 mm.

4. Wood, C. A.: The American Encyclopedia and Dictionary of Ophthalmology, Chicago, Cleveland Press, 1916, vol. 8, p. 6174.

This case illustrates the importance of the examiner's keeping his own eye directly behind the test light in order to avoid error in interpreting the position of the fixational corneal light reflex. In this instance the examiner who keeps his own eye 60 degrees away from the test light is introducing an error of 34°18′ in relation to the true fixational position of the corneal light reflex.

SIZE OF CORNEAL IMAGE IN RELATION TO SIZE OF TEST OBJECT

The size of the corneal light reflex has a direct bearing on the size of the test light. This is of definite practical importance, for when the test light is large the corneal light reflex likewise is large and

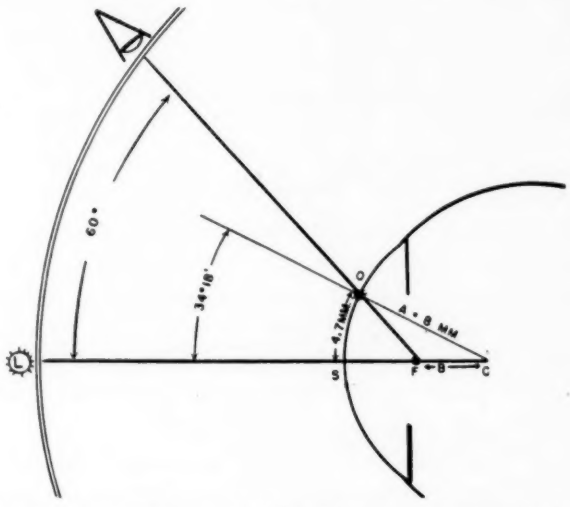


Fig. 5.—Diagram showing how relative positions of the examiner's eye and the test light affect the position of the corneal light reflex.
The diagram is explained in the text.

therefore consumes more of the pupillary area. A corneal light reflex of the smallest possible size to permit critical inspection should therefore prove most satisfactory. Moreover, the light source should be bright, but not so intense as to produce a glaring effect. A medium-sized flashlight with good reflector should prove satisfactory.

Not only is a test light imaged behind the cornea, but one can also observe one's face, reduced in size, and, in the case of a prism over the eye, a reduced image of the prism if one looks between the object prism and the image prism.

The following example will serve to illustrate the relative sizes of object prism and image prism, depending on the distance, as well as the size of said object from the cornea (fig. 4).

A. A prism is placed 10 mm. from the cornea. Where behind the cornea does the image of the prism appear to be?

f = distance from prism to cornea

f' = distance from cornea to image

F = ½ radius of curvature of cornea (8 mm.) = 4 mm.

Formula:

$$\frac{1}{f} + \frac{(-1)}{(f')} = \frac{-1}{F}$$

$$\frac{1}{10} + \frac{(-1)}{(f')} = \frac{-1}{4}$$

$$\frac{1}{f'} = 2.86 \text{ mm. behind anterior surface of cornea}$$

B. What is size of the image of a prism 40 mm. square placed 10 mm. from the cornea?

O = length of prism, or 40 mm.

f = prism to cornea, or 10 mm.

f' = cornea to image, or 2.86 mm. (as determined in preceding formula)

Formula:

$$\frac{I}{O} = \frac{f}{f'}$$

$$\frac{I}{40} = \frac{2.86}{10}$$

$$I = 40 \times \frac{2.86}{10}$$

$$= 11.44 \text{ mm.}$$

DEVIATION OF A CORNEAL LIGHT REFLEX PRODUCED BY A PRISM

It was shown previously that a prism acts much like a mirror. When a mirror is rotated on its vertical axis, the image seen through it can be rectified to an apparently neutral position by interposition of a prism producing an opposite, or neutralizing, effect. What is true of a plane mirror is true also of a curved mirror. Figure 6 shows the parallel relation of the two systems of mirrors. It follows, therefore, that if a prism can rectify the axial displacement of the image in a plane mirror, as well as "correct" the position of the light reflection, it should do the same with a curved mirror. It is this simple displacement which explains the prism reflex test.

Suppose one looks at the image of a point source of light formed either by a plane mirror or by a convex spherical mirror, keeping the eye near the light source. If a prism is interposed in such a way that the image is seen through the prism, the image appears to move toward the vertex of the prism. In the discussion which follows, I shall consider the case in which both the light from the source, and incident on the mirror, and the light reflected from the mirror, and going to the eye, pass through the prism.

One is concerned here with virtual images. A virtual image is defined as one from which the rays of light appear to come. In the figures, the actual paths of the light rays are shown by the solid black lines, and the formation of the virtual images is shown by the interrupted lines. It should be pointed out that a prism will form virtual images only if the vertex angle of the prism and the angular spread

of the cone of rays are both rather small. The diagrams are idealized in that the angular spread of the cone of rays shown is larger than would be permissible. Because of the small angular spread of the cone

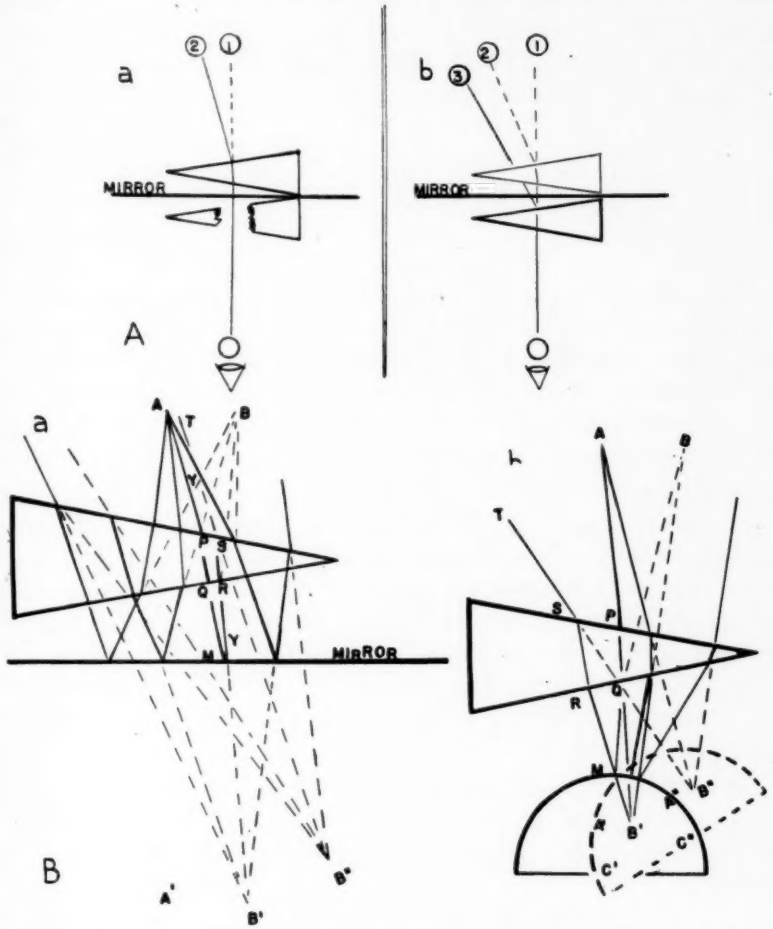


Fig. 6.—The prism reflex phenomenon. *A*, simple schematic drawing. (*a*) Displacement through image prism only. It was shown previously that with any mirror one has a reflection or an image. In this case one has a reflection of both prism and light. It is possible to exclude the image seen through the object prism (as shown in figure 3 *B*) and to study the displacement of light through the image prism alone. The effect as seen in the diagram speaks for itself. The light is displaced toward the apex.

(*b*) Displacements through object and image prisms. The observer looks through the object prism. In doing so, he sees the combined effects of the object and image prisms, respectively. He sees displacement of light from position *1* to position *2*, as in the previous instance, by the image prism; and he also sees the additional displacement of the light from position *2* to position *3* by the object prism. What one sees in making a prism test is the displacement of the image to position *3*. This represents the true strength of the prism, provided it is placed close to the cornea.

B, technical drawing as applied to (*a*) plane mirror and (*b*) convex mirror.

which the eye can accept, there is no difficulty when the image is being seen by the eye. The diagrams are drawn for a 20 D. prism, i. e., one producing an angular deviation of about 11.5 degrees. (If Δ is the power of the prism in diopters, then the angle of deviation is approximately $\Delta/100$ radians, or $1.8\Delta/\pi$ degrees.) For glass, the index of refraction of which is 1.53, this angle of deviation would correspond to a prism angle of about 21 degrees, as shown in the following formula:

n = index of refraction of glass, or 1.53

$1\,\Delta = 0.573°$ (arc)

Formula for using a 20 D. prism:

$$\frac{\Delta \text{ (in degrees)}}{n - 1}$$

$$\frac{20\,(\Delta) \times 0.573}{1.53 - 1} = \frac{11.46°}{0.53°} = 21°$$

The case of reflection from a plane mirror is shown in figure 6 *B*, *a*. The source of light is at point *A*. A pencil of four rays is shown, which, after passing through the prism, appear to come from point *B*. That is, the prism forms a virtual image of *A* at *B*. After reflection from the plane mirror, the rays appear to come from point *B'*, which is the virtual image of *B* formed by the mirror. After passing through the prism a second time, the rays appear to come from point *B''*, which is the virtual image of *B'* formed by the prism. Without the prism, an observer near *A* will see the image of the source at *A'*; with the prism interposed, he will see the image at *B''*. Tracing one of the rays (*AP*) through in detail, one has the following: The ray *AP*, after passing through the prism, follows the path *QM*, which makes an angle, *Y*, with *AP*. After reflection, the path is *MR*, and after the second passage through the prism it is *ST*, which makes an angle, *Y*, with *MR*.

The total displacement of the image by the prism is easily found. If the distance from the light source to the mirror is designated as *D* and the distance from the prism to the mirror as *d*, one has approximately:

$$AB = A'B' = (D - d)\ Y$$
$$B'B'' = (D + d)\ Y$$

and, adding the two equations,

$$A'B'' = 2\ D\ Y$$

It is interesting that—at least to a first approximation—the displacement is independent of the position of the prism, but is directly proportional to the distance of the light from the mirror. The distance *A'B''*, which is the actual linear displacement of the image by the prism, is one which would be hard to measure experimentally except by some sort of parallactic arrangement.

The more important case of a convex spherical mirror is shown in figure 6 *B*, *b*. A pencil of three rays is shown, the middle ray being

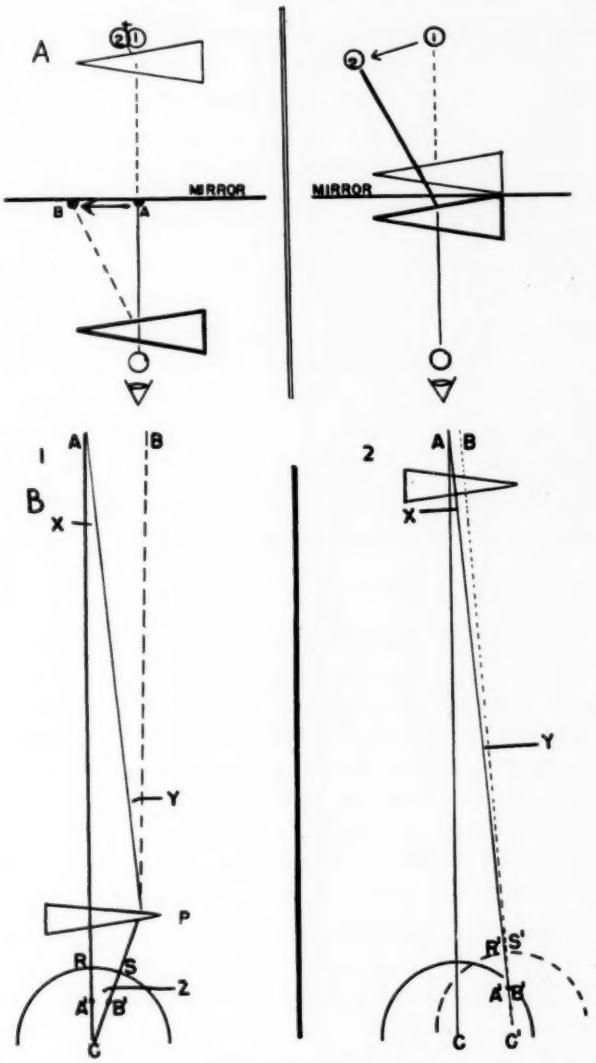


Fig. 7.—Diagrams showing how the distance of the prism from the eye affects prism power, as well as displacement of the corneal light reflex by prism.

A, simple schematic drawing. In this drawing, the prism is placed at two separate distances from the plane mirror—in one instance, at an appreciable distance from the mirror and near the test light, and in the other, close to the mirror. In both instances the image prism assumes positions directly opposite the object prism. In the case in which the prism is placed at an appreciable distance from the mirror, and close to the test light and the observer's eye, one notes a surface displacement of point *A* to point *B* (as in figure 8). This is comparable to seeing the eyeball itself appear shifted, as in the phorometer with the prism drawn away from the eyes. However, since the light reflection is behind the cornea, the "reflex" seen through the mirror at position *1* without prism is shifted through but a small range to position *2*, even though the surface displacement is considerable. The displacement of the light reflex therefore operates inversely to that of the surface shift. When the image prism is close to the image light, the surface displacement is maximal and the light displacement is minimal; when the object prism is close to the surface of the mirror, the light displacement is maximal and represents the true effectiveness of the prism.

B, technical drawing, explained in the text.

the one which strikes the mirror normally and after reflection retraces its path. As before, A' is the virtual image of A, formed by the mirror if the prism is not present; B' is the virtual image of B, which, in turn, is the virtual image of A formed by the prism; C' is the center of curvature of the mirror. The points A'', B'' and C'' are the virtual images of the corresponding points A', B' and C' formed by the prism. An important difference between this case, of the convex spherical mirror, and that of the plain mirror is the fact that in this case one is interested not in the total linear displacement of the image but, rather, in the displacement of the image with respect to the mirror. The reason for this is that since the rays of light by which one sees the mirror also pass through the prism and are bent through the same angle as the reflected rays, one sees a virtual image of the mirror in the position shown by the dotted circle in figure 6 *B*. The final result is that the apparent displacement of the image is $A''B''$, which is the same as $A'B'$, the displacement produced by one passage through the prism.

EFFECTIVENESS OF A PRISM WITH VARYING DISTANCES FROM EYE

It will be noted that as the prism is moved away from the eye and closer to the test light, the eye and the surrounding brow appear to move toward the apex of the prism. This serves to reduce its effective power. The displacement of the light reflex by the prism becomes less as it is moved away from the eye, even though the eye and its surrounding structures, such as the eyelids, appear to move with the rotation of the prism. As the prism is moved closer to the eye, it appears to return (in the direction of the prism base) to its natural position; and when the prism is close to the eyeball, one notes no apparent displacement of surface structures with changing strength of prism. The only effect in this apposed position is in change in the position of the light reflex with variation in the strength of prism. In this apposed position the effectiveness of the prism is maximal.

Figure 7 *B* is a simplified diagram showing only two rays and omitting the prism. The rays shown are $AA'C$, which strikes the mirror normally if the prism is not present, and $APB'C$, which strikes the mirror normally after passing through the prism, to left of P in diagram. The effect of the prism is indicated by the deviation of the ray through angle Y at point P. What is wished is an expression for the displacement of the corneal reflex at the surface of the cornea. In figure 7 *B* this is the distance RS. If D and d have the same meanings as before, then when Y is small, $AC = D$, and $PC = d$, approximately. Applying the "sine law" to triangle APC, one has

$$\sin X = (d/D) \sin Y$$

If the angle Y is reasonably small, one can get a fairly good approximation by setting the sines of the angles as equal to the angles themselves. Doing this, one has

$$X = (d/D)\ Y$$

and, since $X = Y - Z$,

$$Y - Z = (d/D)\ Y$$

One now has for the distance RS:

$$RS = r\ Z$$

$$RS = r\ (1 - d/D)\ Y$$

If the distance from the mirror to the prism is small as compared with the distance from the light to the mirror, then d/D is small as compared with 1, and the displacement, RS, is approximately rY. In other words, if the prism is placed close to the eye, the displacement of the corneal reflex along the surface of the cornea is simply the radius of curvature of the cornea multiplied by the angle of the deviation produced by the prism, this angle being measured in radians, and being equal to the power of the prism, expressed in diopters, divided by 100. On the other hand, as the prism is moved closer to the light, d/D approaches 1 and Z approaches 0.

If point S is the center of the cornea, the rays of light which pass through the cornea will be brought to a focus on the center of the retina. That is, when the reflex is brought to the center of the cornea, the rays will be along the visual axis of the eye. If the prism is close to the eye, the angle of deviation of the eye is exactly the angle of deviation of the prism required to correct it.

In conclusion, as the prism is moved toward the light, the eye (mirror) moves toward the apex of the prism, since the displacement of the mirror by the prism is directly proportional to the distance from the mirror to the prism. At the same time, the displacement of the reflex on the mirror becomes smaller, since the angle Z, which is approximately $(1—d/D)Y$, becomes smaller as d becomes larger and is 0 when $d = D$.

When the prism is moved toward the eye, the image of the eye moves toward the base (of the prism), owing to the fact that as the distance from prism to eye is decreased the displacement of the eye becomes smaller and the eye moves toward its actual position, i. e., toward the base. At the same time, the displacement of the reflex on the mirror becomes larger, since Z increases as d increases.

PRACTICAL CONSIDERATIONS IN THE PRISM REFLEX TEST†

On the basis of the foregoing problems, one must conclude that the prism reflex test is closely related to established methods of prism testing. Because it is objective, it simplifies the clinical use of the

prism. The position of the examiner's eye exactly behind the test light and the placement of the prism close to the eye are important if one is to obtain the optimal value of the prism. Thorington,[5] Hardy[6] and others have shown that clumsy placement of a prism before a patient's eye will yield variable readings for a particular prism strength. The reports are valuable only from an academic standpoint because one would not think of holding a prism in the extreme positions which these authors have shown as leading to greatest errors. They mention the split beam position, the Prentice position and the reverse Prentice position as being nearest the true value of the prism reading. However accurate one may be in the placing of the prism before the eye, one cannot be too exact in attaining either of the aforementioned ideal positions. A commoner reason for error which these authors failed to mention was the abnormally large distance of the rotary prisms from the eyes in the case of the phorometer. Because of the presence of the forehead rest and the frame structure between the prisms and the eyes, producing a separation averaging 4 to 5 cm., one can expect gross inaccuracy in prism readings. If one holds the test light at a distance of 25 cm. from the eyes, the effectiveness of the prism is reduced between 16 to 20 per cent, so that a prism reading of 30△ would really indicate 25 △ or less. For that reason, I prefer the hand rotary prism, such as the Hughes type, which one can place close to the eyes, and which therefore would not affect the numerical reading of the prism to any notable extent.

Another disadvantage of the phorometer is the apparent shift of the eyeball medially as a base-out effect is produced. Considering that the prism may be as much as 5 cm. from the eyes, one would expect displacement of an image or an apparent eye and face displacement of 0.5 mm. for each prism diopter. With a prism of 30 △ base out, the apparent displacement of the stationary cornea would be 1.5 cm., and with 60 △ it would amount to 3 cm., in which case the cornea would be well hidden behind the framework of the phorometer (fig. 8). Here, too, it is the construction of this instrument and the large distance of the rotary prisms from the eye which produce such an effect. This phenomenon is entirely distinct from effective prism power, which was previously discussed. The hand rotary prism placed close to the eyes would obviate such a shortcoming.

Bearing in mind the aforementioned reasons for error with the prism method, as well as the fact that the prism is more accurate in

5. Thorington, J.: Prisms: Their Use and Equivalents, Philadelphia, P. Blakiston's Sons & Co., 1913.

6. Hardy, L. H.: Clinical Uses of Ophthalmic Prisms (Metric), Arch. Ophth. **34**:16-23 (July) 1945.

the lower values and for lesser degrees of squint, the real value of the prism lies in its ease of handling, rather than in reliability in readings. At best, I regard a prism reading as an approximation. In my experience, the anglometer [6a] has proved the simplest and most accurate method of measuring ocular deviations, because it is based on the cover test and on simple adjustment of the lighted target to any position of a deviated visual axis under controlled conditions.

In order to judge the true position of the corneal light reflex, the pupil should not be dilated by any mydriatic. With a small pupil one can make an accurate note of an angle gamma. With a greatly dilated pupil one may be confused as to whether such a light reflex is central or off center.

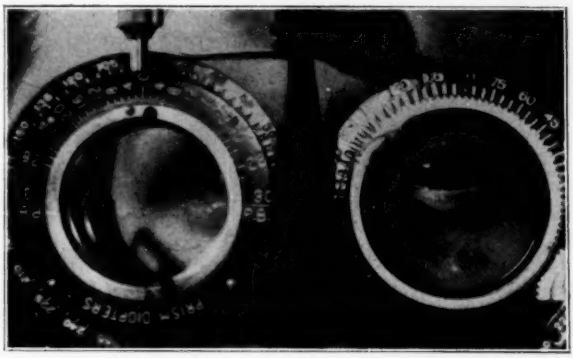


Fig. 8.—Photograph showing how the phorometer reduces the effectiveness of its prisms.

The phorometer has a definite disadvantage over the simple hand rotary prism because of the excessive framework, which increases the distance between the eyes and the prism. In order that any prism may be effective, it should be placed as close to the eyes as possible. Note how the right eye is displaced inward and its cornea is hidden from view by the 30△ base-out prism. This means that the effectiveness of this mechanism is zero because the eye is completely hidden from view.

The prism reflex test is dependable and satisfactory at a working distance of 13 inches (33 cm.) or less. At the near ranges the examiner can keep his eye directly behind the test light and in direct line with the patient's pupil.

For the 20 foot (6 meter) distance, the observation of the corneal light reflexes cannot be controlled as readily as for the 13 inch range, because the test light at that distance is too faint to register a visible corneal light reflex. Moreover, the examiner's head would be in the direct path of the light beam. By using a brighter light, say a 10,

6a. Krimsky, E.: The Cardinal Anglometer, Arch. Ophth. **26**:670-674 (Oct.) 1941.

25 or 40 watt Mazda lamp, for the 20 foot distance, and a magnifying mirror held obliquely in front of the patient's forehead, as in my stereoscope,[7] I have been able to observe the corneal light reflexes clearly

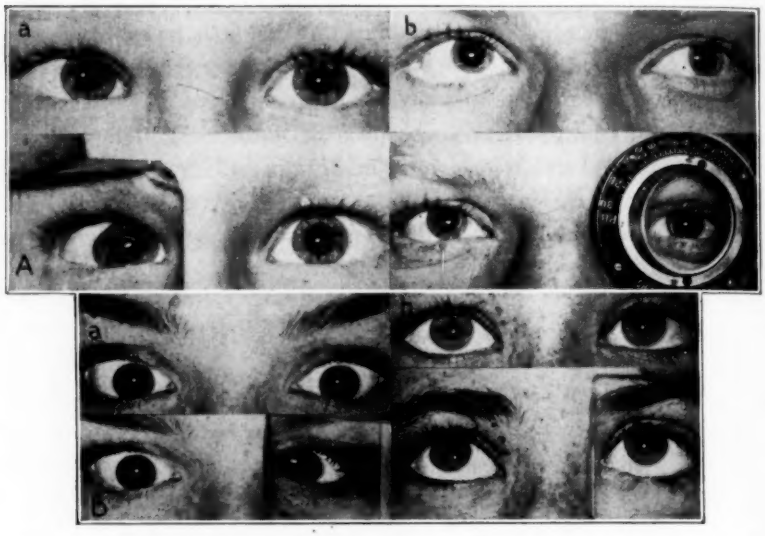


Fig. 9.—Photographic demonstration of clinical applications of the prism reflex test.

A, measurement of heterotropia or manifest squint. (*a*) Convergent squint: In manifest squint the right eye is deviated inward. As a result, the corneal light reflex is displaced outward. A base-out prism of sufficient strength to restore the light reflex in that eye to its fixational position represents the measure of the deviation. A supplementary cover test will confirm the correctness of the prism reflex test.

(*b*) Divergent squint: The left eye is deviated outward and the light reflex is displaced inward. A rotary prism to produce the effect of a 30△ base-in prism (as in the illustration) also restores the light reflex to a central, or fixational, position without changing the position of the eyeball itself. This serves as measure of the deviation.

B, measurement of prism vergence in binocular fixation. (*a*) Prism convergence. First, eyes straight with the light reflexes in their fixational positions. A base-out prism (40△) is then placed over the left eye, and the reflex is still in a fixational position because that eye has converged to overcome the displacement that would otherwise be produced by the prism. The result is the maintenance of binocular fixational reflexes with binocular single vision.

(*b*) Objective diagnosis of heterophoria—exophoria. A "strong" base-down prism is placed over the left eye, as in the familiar prism dissociation, or von Graefe, test, except that the examiner observes the corneal light reflexes at the same time. One now sees that as a result of the prism the light reflexes are no longer fixational in both eyes. The light reflex is in fixational position in the left eye but in the right eye it has shifted downward and inward; the downward component is due to the prism, and the inward component is due to a latent deviation of the outward component, or exophoria. The image response is crossed vertical diplopia, corresponding to the positions of the light reflexes. Measurement consists in transforming this to a true vertical diplopia by supplementing with added base-in prism, which moves the light reflex from an inward to a midline position.

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7. Krimsky, E.: Modification of the Brewster Stereoscope for Clinical Requirements, Arch. Ophth. **26**:808-815 (Nov.) 1941.

through the mirror while standing behind the patient and, at the same time, to perform the cover test satisfactorily.

A small test light at the 20 foot distance would subtend too small an angle to register a light reflex of proper size. One may object to a larger test light because it is too large for critical fixation. In my experience, a small 20 foot test light should be reserved for those who depend on the Maddox rod test to provide subjective information based on a fine streak image. The small 20 foot test light can be dispensed with for the prism reflex test if one is seeking objective information.

In making a prism test, the examiner should look through the object prism rather than above it. As shown previously, the displacement of the light reflex by both the object prism and the image prism represents the power of a particular prism. Instinctively, however, the examiner looks through the object prism without necessarily realizing the true importance of such direct inspection.

CONCLUSIONS

1. The cornea is a convex spherical mirror. Hence, the corneal light reflex is an erect virtual image at the principal focus of the sphere.

2. The corneal light reflex is a misnomer because it is subcorneal in position and is 3 to 4 mm. behind the anterior surface of the cornea. It is technically a reflection.

3. Rotation of a mirror on its edge, or vertical axis, produces a displacement of the lighted image similar to that which one would expect with displacement of the image by a prism.

4. Such a reflex image can be restored to a neutral position by corrective prism. Moreover, the visual axis, which is bent through rotation of the mirror, is also apparently rectified by such prism.

5. The corneal light reflex is, strictly speaking, not a Purkinje image but a light reflection, which one would find in any convex spherical mirror.

6. The prism displaces the corneal light reflex toward its apex in an eye which remains stationary. The amount of prism correction corresponds accurately to the numerical strength of the prism only when such prism is placed close to the eye.

7. The size of the corneal light reflex is important for critical testing. A large reflex may prove confusing.

8. The true measure of the strength of a prism depends on the examiner's looking through the prism rather than above or below it. It is a combination of object prism and image prism which registers

the true value of the prism. If one looks above the prism, one is apt to see only a partial displacement of the light reflex, resulting from the image prism only. In making a routine prism test, one instinctively looks through the prism because it covers so large a surface of the eye.

9. The distance of a prism from the eye has an important bearing on its strength. Undue separation of the prism from the eye reduces its effectiveness by as much as 20 per cent. For that reason, the phorometer is inaccurate. The hand rotary prism or prism rack or single prisms can be handled more effectively.

Miss Sonia Mitchell assisted in the construction and technical description of figure 6 *B*, and Dr. Edwin W. Bechtold reviewed the data after completion of the manuscript.

745 Eastern Parkway.

LIGAMENT OF LOCKWOOD IN RELATION TO SURGERY OF THE INFERIOR OBLIQUE AND INFERIOR RECTUS MUSCLES

WALTER H. FINK, M.D.
MINNEAPOLIS

AN ANATOMIC study of the suspensory ligament of the eye, (ligament of Lockwood) based on a series of dissections, is presented. Certain phases of the anatomic structure of the ligament which seem to be of importance in the management of abnormalities of the inferior oblique and inferior rectus muscles are presented.

The suspensory ligament, as described by Lockwood, consists of a blending of the sheaths of the inferior oblique and inferior rectus muscles to form a special thickening of the fascial structure. The lateral expansion of this sheath extends upward laterally and medially to join the sheaths of the lateral and medial rectus muscles, thereby gaining indirect attachment to the orbital margin. There is thus formed a continuous band about 0.1 inch (0.25 mm.) thick beneath the globe, which supports it like a hammock.

Generally speaking, the ligament is thought of as a "suspensory hammock" which functions as a support to the eyeball. Insufficient emphasis has been placed on its other functions, which have to do with its relation to the inferior oblique and inferior rectus muscles. From a review of the literature, supplemented with a series of dissections, it is apparent that, in addition to the suspensory action, the ligament has a definite function in promoting a smooth, integrated action of these muscles. In the region of the inferior oblique and inferior rectus muscles, the structure of the ligament becomes modified, thus enabling it to perform certain specialized functions.

The investigation emphasized three important modifications of the ligament in its relation to these extraocular muscles. First, and perhaps most important, is the modification of the ligament at the point of crossing of the two muscles. Second is the modification of the ligament which serves as a sheath to the inferior oblique muscle lateral to the point of crossing. Third is the modification of the ligament which functions as a check ligament to the inferior oblique muscle, and, in addition, acts in conjunction with the inferior oblique muscle to serve as a check ligament to the inferior rectus muscle.

The most important modification of the suspensory ligament is found

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at the point of crossing of the inferior rectus and the inferior oblique muscle. Various descriptions of this anatomic structure are found in the literature, but little emphasis has been placed on the variations which may be present in this highly specialized area, and insufficient stress is placed on its possible clinical importance.

A study of anatomic material reveals that a firm union exists between the inferior oblique and the inferior rectus muscle. This is produced by a fusion of the fascial sheaths of these muscles at the point of crossing. The fascial sheath on the under surface of the inferior rectus muscle becomes definitely thickened a few millimeters before it reaches the inferior oblique muscle. At the point where the muscles cross, this thickened fascia comes in contact with a similar thickening of the fascia

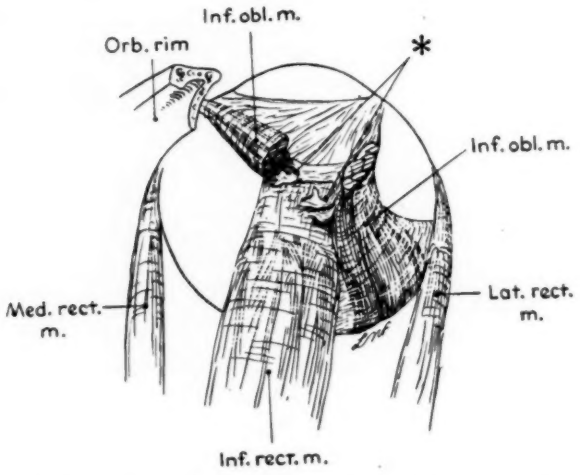


Fig. 1.—Crossing of the inferior oblique and the inferior rectus muscle as seen from below. The inferior oblique muscle is severed and turned laterally, showing a firm adhesion between the fascial sheaths of the two muscles. The asterisk indicates short, thick adhesions between the two muscles, which would cause practically complete fusion between the sheaths, resulting in little, if any, independent action of the two muscles.

covering the inferior oblique muscle. A fusion is brought about by numerous fascial bands which pass from one fascial sheath to the other.

Study of a series of specimens revealed that the degree of fusion between the fascial sheaths of these muscles is variable. Some specimens showed a firm union, which seemed to be a complete fusion of the fascial sheaths (fig. 1). Others showed a very loose fusion, with but a few bands of fibrous tissue connecting the two fascial sheaths, and no difficulty was experienced in separating them (fig. 2). Between these two types there was a series of specimens in which a fairly firm union existed and the fusion of the sheaths was moderate in degree. In these specimens

the fascial strands, although firmly uniting the two muscle sheaths, did not produce their complete fusion.

The degree of independent movement of each muscle was in proportion to the degree of fusion of the fascial coverings of the two muscles. In specimens showing a firm union, there did not seem to be any apparent independent movement of the two muscles, and they gave the impression of a solidly fused structure. In specimens with loose fusion, the independent movement of the two muscles was definite. As would be expected, the specimens between these extremes showed variable amounts of movement, and the movement was in proportion to the degree of fusion present. The majority fell into the latter category.

It could be demonstrated that in the region where the muscles crossed the thickened fascial covering of the muscles was firmly adherent

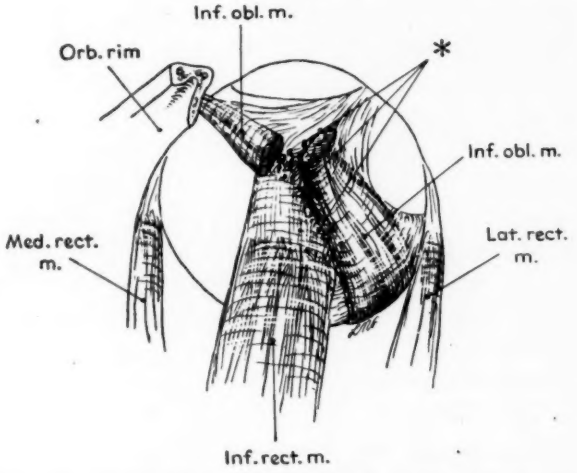


Fig. 2.—Crossing of the inferior oblique and the inferior rectus muscle as seen from below. The inferior oblique muscle is severed and turned laterally, showing loose adhesions between the fascial sheaths of the two muscles. The asterisk indicates long, delicate, loose bands of fibroelastic tissue, permitting considerable independent action of the two muscles.

to the muscle tissue. Whereas the outer layer was largely made up of circular fibers, the inner layer, which was in contact with the muscle tissue, had fibers which were mostly longitudinal. The inner layer was apparently continuous with the internal perimysium of the muscle and caused the muscle fibers to be firmly united to the fascial covering of the muscle.

It is obviously impossible to compare exactly prepared anatomic material with a similar structure in the living tissue. Anatomic material should, however, provide a basis for comparison of different degrees of union, because this variable degree of fusion of the structures should have much the same relation as in the living tissue.

From a clinical standpoint, it would seem that because of such an intimate fusion of the two muscles, any surgical manipulation of these muscles should be evaluated with this anatomic fact in mind. Such a variation in degree of independent action of the muscles should to some degree influence the functioning power of the muscles. The influence should be especially evident when an operation is performed on the muscle, particularly at the origin of the inferior oblique muscle. Operation at the origin of this muscle should produce variable results because of the variations in the degree of fusion at the point of crossing to the two muscles. It is logical to assume that a firm fusion between the muscles should lessen the effect of a tenotomy at the origin, more than would a fusion of less degree. One may conclude, herefore, that in surgical treatment of the inferior oblique, the insertion, rather than the origin, of the muscle is the logical point for the surgical approach, because more definite results may be anticipated.

The degree of fusion of the fascial sheaths at the crossing of the muscles may in a measure influence, likewise, the result when operation is performed on the inferior rectus muscle, because the fascial structure at this point likewise influences the action of this muscle.

One is tempted to speculate that a pronounced variation in the arrangement of the fascial tissues at this point might influence the physiologic action of these muscles to such a degree that clinical results commonly referred to as paresis or overaction of one or the other muscles would be produced.

It should be kept in mind that in operations on the inferior oblique muscle, such as a recession, the new point of attachment must not deviate from the plane of action of the muscle. The fixed position of crossing of the muscles demands this in order that the muscle may function efficiently. The question of the constant location of this point of crossing does not seem to be significant. The measurements taken in 40 specimens showed much the same relation to the eyeball.

The second anatomic consideration of the suspensory ligament of the eye is the modification of the ligament which forms a sheath for the inferior oblique muscle lateral to the point of crossing. As was true in the case of the sheath at the point of crossing of the muscle, the sheath in this area also had two layers, an outer layer with circular fibers predominating, and the inner layer, with longitudinal fibers predominating. The inner layer was continuous with the muscle tissue, being united with bands which blended with the perimysium of the muscle. These bands are of special significance, for they form an intimate connection between the muscle tissue and the muscle sheath, and at

operation it is occasionally necessary to separate the muscle from its sheath in order to obtain a satisfactory result.

The nerve and blood supplies to the muscle perforated the sheath on the posterior edge just after it crossed the inferior rectus muscle. Near the insertion, the muscle sheath fanned out anteriorly to fuse with the sheath of the lateral rectus muscle and posteriorly to fuse with the fascial sheath surrounding the optic nerve (fig. 3). In this posterior area, the fascia was perforated by the ciliary vessels and nerves.

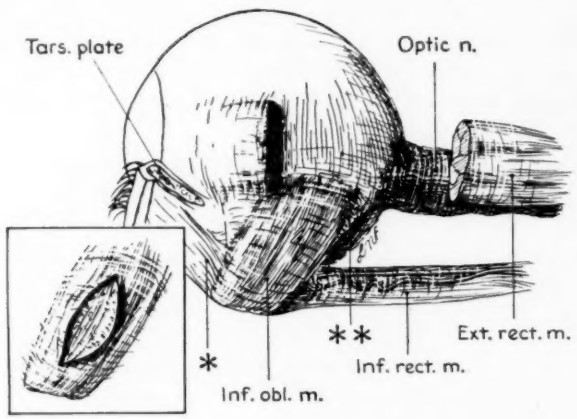


Fig. 3.—Lateral view of the eye, showing the lateral half of the inferior oblique muscle (after its crossing with the inferior rectus). The drawing shows the extensions of the membranous sheath covering the inferior oblique muscle anterior to the sheath of the lateral rectus muscle and posterior to the fascia surrounding the optic nerve. It is particularly important that these fibrous extensions be severed in doing operative work on the inferior oblique muscle.

The single asterisk indicates the fascial sheath extending forward to the tarsal plate, which acts as a check ligament to the inferior oblique muscle. The double asterisk indicates the posterior extension of the sheath of the inferior oblique muscle to fuse with the sheath of the optic nerve.

The small insert illustrates a portion of the inferior oblique muscle with its fascial sheath. In this drawing, a portion of the sheath has been cut to permit a view of the fibrillae which pass from the inner surface of the sheath to the perimysium of the muscle, thus forming a firm union between the sheath and the muscle.

It is obviously important to sever all of this sheath, as well as the insertion of the muscle, in order to secure the desired surgical effect. To sever only the muscle and do a recession while the sheath remains intact at the posterior edge would tend to nullify the effect desired. In severing the sheath, it is important to remember its relation to the ciliary vessels and nerves in this region—the fovea, which is just above the posterior tip of the muscle; the vortex vein, and the artery and nerve

supplying the muscle (fig. 4). It is well to also bear in mind the firm union of the fascial sheath and the muscle.

The third anatomic consideration of the suspensory ligament of the eye is the modification, which acts as a check ligament to the inferior oblique muscle. Motais [1] was the first to demonstrate this function of the suspensory ligament, and Maddox [2] gave the following description:

" . . . a fibrous bundle, derived in part from the fibres of the sheath of the inferior rectus, in part from the sheath of the inferior oblique muscle, which leaves the anterior border of the inferior oblique about 8 or 10 millimeters from its orbital origin, and from thence courses obliquely outwards and forwards. It forms an obtuse angle of about 120° with the check ligament of the inferior rectus muscle. With the inferior oblique it forms an angle of about 110°. Its length is from 10 to 12 millimeters, and it is inserted into the lower outer angle of the orbit,

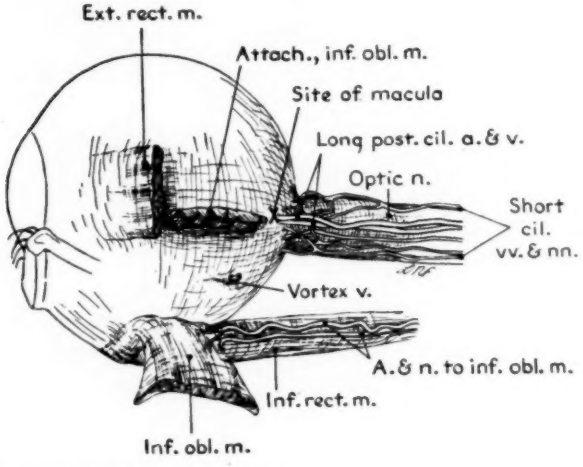


Fig. 4.—Lateral view of the eyeball, in which the inferior oblique muscle is cut and turned downward to show its line of insertion to the sclera. The figure, although diagrammatic, shows accurately the relations of the various structures as observed in anatomic specimens.

The vortex vein is usually seen 10 to 12 mm. below the posterior end of the attachment of the inferior oblique muscle and immediately under the posterior border of the muscle. The diagram attempts to illustrate the oblique position of the vortex vein as it enters the sclera. Note the relation of the macula, ciliary vessels and nerves to the posterior tip of the insertion of the inferior oblique muscle. The artery and nerve enter the inferior oblique muscle just lateral to the point where this muscle crosses the inferior rectus muscle.

4 or 5 millimeters behind the orbital margin, about midway between the external check ligament and the origin of the inferor oblique.

This bundle is mostly pearly looking, the most purely fibrous of all the aponeurotic lamellae of the orbit.

1. Motais, E.: Recherches sur l'anatomie humaine et l'anatomie comparée de l'appareil moteur de l'oeil, Arch. d'opht. **4**:532-537, 1885.

2. Maddox, E. E.: Tests and Studies of Ocular Muscles, Bristol, J. Wright & Co., 1898, p. 32.

Its breadth varies at different parts of its course: in the middle, 2 or 3 millimeters; at its muscular insertion, 7 or 8 millimeters; at its bony insertion, 5 or 6 millimeters. It presents, therefore, the shape of two triangles united by their apices. Together with the inferior oblique itself, it forms a kind of musculo-aponeurotic loop, the two ends of which are inserted near the orbital margin, one at the outer angle, the other at the inner angle. The check ligament of the inferior rectus muscle embraces the middle portion of this loop, so that when the inferior rectus begins to contract, its check ligament stresses the loop.

Duke-Elder [3] mentioned the presence of a fascial strand running laterally and backward to the orbital floor. Others have expressed doubt, however, as to the existence of a definite check ligament of the inferior oblique muscle. Whitnall [4] pointed out that in his dissections he found

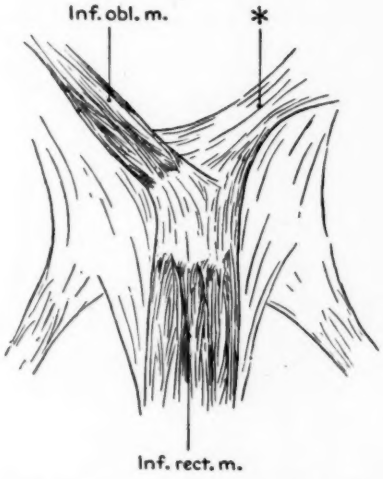


Fig. 5.—Diagram taken from the work of Motais. The asterisk illustrates the arrangement of the check ligament of the inferior oblique muscle. According to Motais, this check ligament, together with the inferior oblique muscle, has a check action on the inferior rectus muscle. The arrangement is described as a Y-shaped affair. With such an arrangement, it is obvious that a tenotomy of the inferior oblique at its origin would affect the check action on this muscle and likewise affect the check action on the inferior rectus muscle.

but 1 instance in which "a strand . . . would be reasonably possessing any such action." Fuchs [5] stated that "the inferior oblique has no check ligament, its place being taken by the ligament attached to the reflected tendon of the superior oblique."

3. Duke-Elder, W. S.: Text-Book of Ophthalmology, St. Louis, C. V. Mosby Company, 1933, vol. 1, p. 181.

4. Whitnall, S. E.: Anatomy of the Human Orbit, New York, Oxford University Press, 1932, p. 297.

5. Fuchs, E.: Textbook of Ophthalmology, translated by A. Duane ed. 7, Philadelphia, J. B. Lippincott Company, 1923, p. 58.

With such a difference of opinion, the question arises as to the exact status of the check ligament of the inferior oblique. It would seem that the ligament must exert some check, but it is doubtful whether it is the well defined mechanism described by Motais.

In the present series of dissections, an effort was made to answer the question. After careful investigation, it seems apparent that no such mechanism as that described by Motais was evident in this series of specimens. It could be demonstrated that in the location described there were present bands of fascia in various numbers, and arranged in such a manner that they could act in the capacity of a check ligament to the inferior oblique muscle. Most of these strands blended posteriorly with the fibrous sheath of the inferior oblique, and anteriorly seemed to be attached to, or blended with, the septal membrane of the orbit, thus

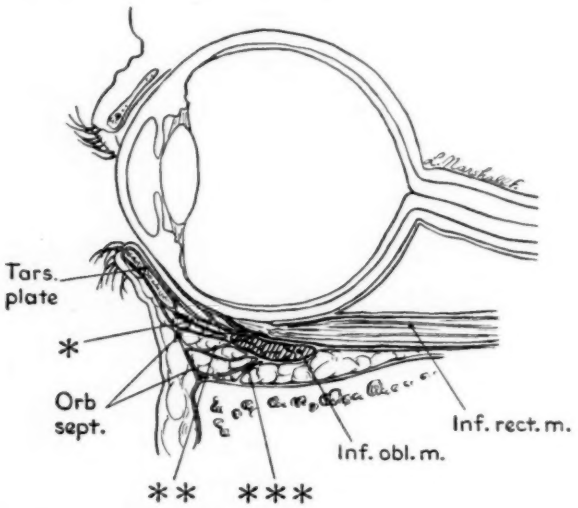


Fig. 6.—Vertical section of the check ligament of the inferior oblique muscle. The fascial covering of the inferior oblique muscle extends forward, having extensions to the lower cul-de-sac, the lower tarsal plate and the septum orbitale, and a few strands to the periosteum on the floor of the orbit.

The single asterisk indicates fibrous bands extending to the tarsal plate; the double asterisks, fibrous bands extending forward to the septum orbitale, and the triple asterisks, fibers extending to the periosteum on the floor of the orbit. These are usually not direct extensions but are made up of the fibrous network which surrounds fatty tissue.

becoming indirectly attached to the orbital rim. Other strands seemed to blend with the supporting structure surrounding the fatty tissue, and were thus indirectly attached to the periosteum of the floor of the orbit immediately behind the orbital rim (fig. 6). In no case was a definite ligament present, as described by Motais; instead, the arrangement appeared rather as a broad meshwork of fascial fibers, which in some instances was compact and had the appearance of an extension of the muscle sheath.

This fascial membrane in several cases was definitely more compact near the point of crossing of the muscles and formed a firm band between this area and the orbital septum immediately anterior to it (fig. 7). Although the arrangement was not that described by Motais it is reasonable that such a structure could act as a check ligament to the inferior oblique muscle and, through its action with the inferior oblique, as check ligament to the inferior rectus muscle.

As in the case of the other check ligaments, this structure undoubtedly is a factor in promoting more efficient action of the inferior oblique muscle. Motais expressed the belief that it acts not only as a moderator of the action of the inferior oblique, but also as a "pulley of reflection." By this he must have meant that when the muscle contracts the ligament

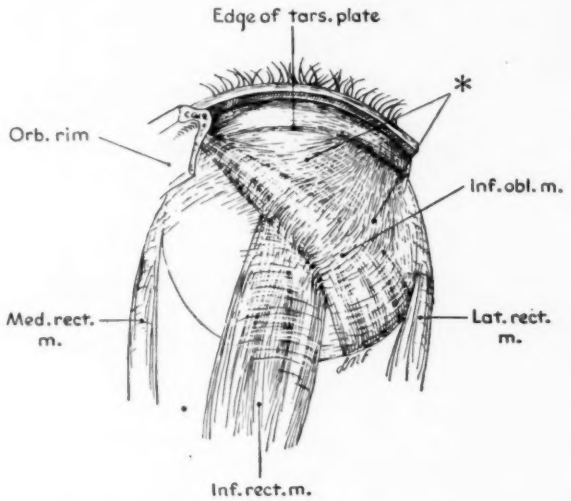


Fig. 7.—Fascial arrangement covering the inferior oblique and inferior rectus muscles as seen from below.

The asterisk illustrates how the fascial covering of the inferior oblique muscle is continued forward and fuses with the lower edge and with the body of the tarsal plate. It will be noted that in the region of the crossing of the muscles the membrane is denser and takes on somewhat the arrangement described by Motais, although it is not so sharply defined. By this specialized arrangement the ligament of Lockwood serves as a check ligament to the inferior oblique muscle and, in conjunction with this muscle, acts as a check ligament to the inferior rectus muscle.

slightly bends the muscle by drawing its middle part outward, so as to make its traction on the eye a little less oblique.

A surgical approach to the inferior oblique muscle should avoid interference with the action of this structure. Tenotomy performed on the insertion would be less likely to interfere with its action than would an operation at the origin of the muscle.

The check ligament of the inferior oblique muscle very likely plays an important role in conjunction with the inferior oblique muscle to act with the check ligament to the inferior rectus muscle. The check ligament of the inferior rectus apparently has no direct insertion into the margin of the orbit, but acts only through the aforementioned structure, one limb of which is formed by the inferior oblique muscle and the other by the check ligament of the same muscle. As stated by Motais,

> The check ligament of the inferior rectus has, therefore, for its orbital insertions, the tendon of the inferior oblique muscle, and the check ligament of the same muscle like the two limbs of a Y.

If such an anatomic arrangement is present, it is evident that tenotomy of the inferior oblique at its origin will also disrupt a balanced check action on the inferior rectus muscle and thus cripple the normal action of that muscle. This factor alone should cause one to avoid any surgical manipulation of the inferior oblique muscle at its origin.

It is also important to emphasize that an extension of the suspensory ligament of the eye also is attached to the lower tarsal plate and cul-de-sac and tends to hold them in place. Surgical manipulation of the inferior rectus should be such that this extension is not disturbed.

CONCLUSION

The suspensory ligament of the eye (ligament of Lockwood), in addition to being a "suspensory hammock," is of importance to the clinician because of its specialized functions in relation to the inferior oblique and the inferior rectus muscle.

From an anatomic standpoint, the surgical approach to the inferior oblique muscle should be at its insertion. Operation at the origin of the inferior oblique would produce more uncertain results because of the variable degree of fusion with the inferior rectus muscle at the point of crossing and would also tend to disturb the action of the check ligament, which is essential to an efficient action of the muscle. Operation at the origin of the inferior oblique muscle would also weaken and distort the check action on the inferior rectus muscle, an action which is dependent on a balance of pull between the inferior oblique muscle, from its crossing to its origin, and the check ligament of the inferior oblique muscle.

In operation at the insertion of the inferior oblique, one should consider not only the insertion of the muscle but also the insertion of the muscle sheath and the important structures passing through it. In recession, the muscle should be reattached in the line of action.

It is important, therefore, in contemplating any surgical procedure on the ocular muscles, to evaluate the step with the anatomic facts in mind. An ill advised surgical procedure not only may be ineffectual but may also cripple the action of more than one muscle.

1029 Medical Arts Building.

ABSTRACT OF DISCUSSION

DR. MAYNARD WHEELER, New York: We are greatly indebted to Dr. Fink for making this study. Most physicians think that they are too busy to go back to the cadaver for review, not to mention new discoveries. Fortunately, there are a few staunch members of the medical profession who are willing to make this sacrifice. The recent trend in surgery of the inferior oblique has been to shift attention from the origin to the insertion of this muscle. Dr. Fink has now placed this on a rational basis. However, at the same time, he has added to the already formidable list of hazards to be encountered in this region. He has explained the highly variable results that all have experienced in doing myectomies near the origin of the inferior oblique muscle. No doubt, in those embarrassing cases in which some overaction of this muscle persists after a myectomy a firm union between the inferior oblique and the inferior rectus muscle existed. Dr. Fink hinted at the possibility that this firm union might explain the overaction of the inferior oblique muscle which so frequently accompanies lateral squint. I wish that he would speculate further on this important point. If the two inferior muscles are firmly adherent to their sheaths and the sheaths are closely united, it is hard to see how these somewhat antagonistic muscles function properly and independently.

Dr. Fink mentioned that Lockwood's ligament forms a suspensory hammock to support the eye and that the ligament extends to fuse with the fascia of the lateral rectus muscle on one side, and with that of the medial rectus muscle on the other. It has always been my custom, in operating on the medial and lateral rectus muscles, to cut freely Tenon's capsule along the lower margins of these two muscles. I have never been aware of any dropping of the globe, but possibly I have missed it. I should like to know what Dr. Fink thinks about this.

DR. HAROLD F. FALLS, Ann Arbor, Mich.: Dr. Fink's meticulous anatomic research and carefully weighed conclusions deserve the grateful thanks of all ophthalmic surgeons. The late Dr. White, of New York, was untiring in his efforts to point out that a greater effect could be obtained from tenotomy of the inferior oblique muscle at its insertion than at its origin. The anatomic explanation of this observation has, conceivably, been furnished by Dr. Fink in his demonstration of the frequent firm union between the fascial sheaths of the inferior rectus and the inferior oblique muscle. Such fusion of the fascial sheaths could well serve as a new site of origin for the inferior oblique muscle after myectomy or tenotomy at its origin, and may thus account for frequent poor, inadequate results. It is unfortunate that there are not available adequate or specific clinical signs which would permit one to judge before operation whether or not a firm union is present. With respect to surgical intervention on the inferior oblique muscle, I feel strongly that a recession of the muscle at its insertion is to be preferred to a myectomy or a tenotomy performed at either the origin or the insertion. There is no more excuse today for performing a tenotomy of the inferior oblique muscle than one of the inferior rectus. Experience has taught that a carefully placed recession is to be preferred, for if it is necessary to correct or to increase the result, little difficulty is encountered in again locating the muscle. If, however, the surgeon prefers tenotomy, I urge that it be done at the insertion, for, again, it is technically simpler

and can be corrected if overaction results. In my hands, myectomy at the site of origin has been a poor surgical procedure, for it is technically unsound and its effects are permanent and unalterable. Dr. Fink points out that "it is important to sever all of the inferior oblique sheath, as well as the entire insertion of the muscle," and in this statement I concur.

I am pleased that Dr. Fink has emphasized the importance of the check ligament of the inferior rectus muscle, especially with reference to its attachment to the tarsus of the lower lid and the conjunctiva of the lower cul-de-sac. This relation must be kept in mind by the surgeon when attacking the inferior rectus, for excessive recession of this muscle will pull down the lower lid and effect a widened palpebral fissure. It is equally true that excessive resection of the same muscle will narrow the vertical width of the palpebral fissure. Duane was aware of this and pointed out that a recession of the inferior rectus should not exceed 3 mm. and that a resection should be limited to 5 mm. Dr. F. B. Fralick, in his Academy course on ocular anatomicosurgical principles, points out that most visual activity lies well below the horizontal plane, and thus falls into the sphere of action of the inferior rectus muscle. The surgeon is cautioned to avoid any operative procedure that would cripple the action of this muscle. Dr. Fink also cautions the surgeon to evaluate any surgical procedure with all the anatomic facts in mind for any ill advised procedure not only may be ineffectual but may cripple the action of more than one muscle. When conclusions based on both clinical and research experience are so similar, it certainly behooves the ophthalmic surgeon to be more careful in his choice of the operative procedure in each case.

Dr. Walter H. Fink, Minneapolis: In his daily contact with problems of the extraocular muscles, the ophthalmologist occasionally encounters cases in which an oblique muscle is responsible for the defect. The management of such cases has caused me concern because of my uncertainty as to the proper procedure. This is especially so in considering surgical procedures on this muscle. From observations in a series of dissections of these muscles, it is evident that effective surgical correction can be accomplished only when the operator is thoroughly familiar with the anatomy of the area. It is particularly important that he be aware of the anatomic variations of the muscles. It may be mentioned in passing that in some cases such anatomic variation may be the important factor in abnormal action of the oblique muscles. Another factor which should receive special emphasis is the surgical approach to the inferior rectus muscle. Many operators believe that operation on this muscle should be avoided whenever possible. My dissections bear out this view, for surgical manipulation of this muscle involves disruption of the fascial attachments in this region, which are important in the check action not only of the inferior rectus but also of the inferior oblique muscle.

Clinical Notes

USE OF COATED LENSES IN RETINOSCOPY

GEORGE S. ZUGSMITH, M.D.
SAN PEDRO, CALIF.

RECENT experience in reduction of reflection on coated camera and spectacle lenses suggested the use of coated trial case lenses [1] in retinoscopy. The earlier work on coating of lenses, according to Graham,[2] was done by Dennis Taylor, in England, as early as 1892. He observed that the layer of dust or discoloration which appeared on old camera lenses did not reduce the light they transmitted but tended to increase it. It also reduced reflection. Taylor endeavored to reproduce the effect of age but was unable to get a coating with a wavelength necessary to intercept the incident light so as to counteract its effect. However, Strong, at the California Institute of Technology, and Cartwright and Turner, at the Massachusetts Institute of Technology, developed a process

Fig. 1.—Alterations in wavelength of rays of light *A*, *B* and *C* produced by films of various optical thicknesses.

which was used extensively in World War II on optical instruments and camera lenses. This process is the coating of the finished spectacle lens with a 0.000004 inch (0.0001 mm.) thickness of a hard, inert mineral salt, usually magnesium fluoride, evaporated at high temperature and fused to the surface of the glass under a vacuum of 1/10,000,000 atmosphere. The molecular vapor thus produced condenses on the optical element to form a fairly durable and transparent layer. The coating may be applied to crown or flint glass. On crown glass there is a 6 per cent increased transmission of light, which has made the process useful for camera lenses. At the same time, intensity of light reflected by a treated lens is reduced by about 60 per cent. This is the best that can be obtained with present coatings. In order to produce zero reflection of light, the film must equal in optical thickness one fourth of a wavelength of the incident light, and the refractive index of the film must be a geometric mean of the indexes of the mediums—in this case air and crown glass. If this equation were possible, light ray *A* in the diagram (fig. 1) would be

1. Suggested by Mr. J. Wetmore.

2. Graham, R.: Reduction of Reflection, Arch. Ophth. **36**:315-320 (Sept.) 1946.

destructively interfered with by ray *B*, which is one-half wavelength out of phase, having passed the coating one-fourth wavelength in depth, as seen in the dotted line. If the coating were one-half wavelength thick, there would be an alteration of a full wavelength, as in light ray *C*, and thus the reflected light would be accentuated, as seen in the broken line.

It has also been shown in spectacle lenses that the coating of magnesium fluoride reduces "ghost images" due to interface reflections and gives an increase in contrast and detail. It was felt, therefore, that some of the "shadows" associated with retinoscopy might be in the class of "ghost images" and that the use of coated lenses might eliminate these to a large extent. Through the courtesy of the processing company,[3] a trial lens case was coated and used in this preliminary study of 100 patients. The data in the table are representative of the group.

It was hypothesized that there might be a closer relation between the retinoscopic refraction with coated lenses and the trial case acceptance.

Fig. 2.—Coated (left) and uncoated (right) trial case lenses.

This was found to be true, especially with respect to the cylindric correction and the selection of axis. Retinoscopic studies were made with coated lenses and on the same patients with uncoated lenses as a control.

However, more apparent than the statistical results was the ease with which the retinoscopic examination was performed. The shadow was much clearer and sharper with the coated lenses. The reversal point was more easily seen. There was absence of annoying side images, and in this series no scissors movement was noted in the coated lens, whereas 1 patient was seen to have such a reflex with the uncoated lenses. This aspect requires more study. There is an apparent effect of the trial frame with no lenses in it. Subjectively, the patients stated that retinoscopic examination was not as annoying through the coated lenses as through the uncoated lenses, since the brightness of the retinoscope light could be reduced. Retinoscopic examination through the undilated pupil was easier through the coated lens. In several after-

3. Lane Wells Company, Lester-Lite Division, Pasadena, Calif.

cataract cases the end point was seen clearly and distinctly with a minimum of distortion. The same effect was noted in several high cylindric corrections.

Data from Retinoscopic Study of Seventeen Patients with Use of Coated and Uncoated Trial Case Lenses

Case		Retinoscopic Refraction: Coated Lenses	Retinoscopic Refraction: Uncoated Lenses	Trial Case Acceptance
1	OD:	−0.50 ⌒ +0.25 × 90	−0.25 ⌒ +0.25 × 90	−0.75 ⌒ +0.25 × 90
	OS:	+0.50 ⌒ +0.75 × 75	+0.25 ⌒ +1.25 × 90	+0.25 ⌒ +0.75 × 75
2	OD:	+0.25 ⌒ +0.25 × 90	+0.75 × 90	+0.50
	OS:	+0.25	+0.50	+0.25
3	OD:	−1.00 ⌒ +2.00 × 70	−1.25 ⌒ +2.25 × 80	−1.00 ⌒ +2.00 × 70
	OS:	−1.00 ⌒ +2.25 × 95	−1.25 ⌒ +2.00 × 90	−1.00 ⌒ +2.25 × 100
4	OD:	−0.25 ⌒ +1.50 × 90	−0.25 ⌒ +2.00 × 75	−0.50 ⌒ +1.50 × 90
	OS:	+0.50 ⌒ +0.50 × 75	+0.75 ⌒ +0.50 × 90	+0.25 ⌒ +0.50 × 75
5	OD:	+0.25 ⌒ +0.50 × 105	+0.25 ⌒ +0.50 × 105	+0.50 × 105
	OS:	+0.25 ⌒ +0.25 × 75	+0.50	+0.25 ⌒ +0.25 × 75
6	OD:	+0.25 ⌒ +1.25 × 180	+0.50 ⌒ +1.25 × 180	+0.25 ⌒ +1.50 × 180
	OS:	+0.25 ⌒ +1.50 × 165	+0.25 ⌒ +1.00 × 165	+1.75 × 165
7	OD:	+0.50 ⌒ +0.75 × 90	+0.75 ⌒ +0.75 × 90	+0.25 ⌒ +0.75 × 90
	OS:	+0.25 ⌒ +0.75 × 90	+0.25 ⌒ +0.75 × 90	+0.25 ⌒ +0.75 × 90
8	OD:	+1.00 ⌒ +0.25 × 70	+1.25 ⌒ +0.25 × 90	+1.00 ⌒ +0.25 × 75
	OS:	+1.25 ⌒ +0.25 × 90	+1.50 ⌒ +0.25 × 90	+1.00 ⌒ +0.25 × 90
9	OD:	+1.75	+2.00	+1.50
	OS:	+2.25 ⌒ +0.50 × 160	+1.50 ⌒ +0.75 × 180	+2.00 ⌒ +0.50 × 165
10	OD:	+0.50 ⌒ +0.50 × 105	+0.25 ⌒ +0.75 × 90	+0.75 ⌒ +0.50 × 90
	OS:	+1.75 ⌒ +0.25 × 90	+1.50 ⌒ +0.25 × 90	+2.00
11	OD:	+0.50 ⌒ +0.25 × 90	+1.25 ⌒ +0.25 × 90	+0.25 ⌒ +0.50 × 90
	OS:	+0.75 1.00	+1.00	+0.50
12	OD:	+1.00 ⌒ −1.00 × 90	−0.75 ⌒ −1.00 × 90	−0.87 ⌒ −1.25 × 90
	OS:	−1.00 ⌒ −1.50 × 75	−0.75 ⌒ −1.50 × 75	−1.00 ⌒ −1.50 × 75
13	OD:	−0.25 ⌒ −0.50 × 120	−0.25 ⌒ +0.25 × 15	−0.50 ⌒ −0.25 × 120
	OS:	−1.00 ⌒ +2.00 × 170	−0.75 ⌒ +2.00 × 180	−1.25 ⌒ +2.00 × 165
14	OD:	+1.75	+1.75	+1.50 ⌒ +0.25 × 180
	OS:	+1.50	+1.75	+1.25 ⌒ +0.25 × 165
15	OD:	+1.00 ⌒ −5.50 × 5	+1.25 ⌒ −5.00 × 180	+0.75 ⌒ −5.50 × 5
	OS:	+1.25 ⌒ −4.25 × 170	+1.25 ⌒ −4.50 × 180	+1.50 ⌒ −4.50 × 172
16	OD:	+10.50 ⌒ +3.00 × 30	+11.00 ⌒ +3.50 × 45	+10.00 ⌒ +3.00 × 30
17	OD:	+9.50 ⌒ +6.00 × 15	+10.50 ⌒ +4.00 × 15	+9.50 ⌒ +5.50 × 15

SUMMARY

A series of 100 patients were given retinoscopic examinations through coated and uncoated trial case lenses. There was closer correlation between trial case acceptance and retinoscopic corrections with coated lenses. The retinoscopic shadow was clearer, and side shadows were eliminated. The retinoscopic end point was easier to see. There was less complaint from the patient of discomfort due to brightness of the light.

529 West Eighth Street.

Obituaries

ERNEST FREDERICK KRUG, M.D.
1877-1948

Ernest Frederick Krug died in New York on Feb. 28, 1948. He was born in Cleveland, Ohio, on Oct. 27, 1877, the son of Professor Joseph Krug, who for many years supervised the teaching of German in the public schools of that city. He was graduated from the College of Physicians and Surgeons of Columbia University in 1900. After finishing his general internship at the Lenox Hill Hospital, then known as the German Hospital, he went to Berlin and Vienna for the purpose of equipping himself to practice ophthalmology and otolaryngology. His strict German training and his knowledge of that language stood him in good stead throughout his professional career.

Returning to New York he completed an exceptionally thorough preparation as office assistant to Dr. Emil Gruening. Over a long period he was on the staff of the New York Eye and Ear Infirmary, serving as senior assistant surgeon in the clinic of Dr. Wilbur Marple. During the past twenty-five years he had devoted himself solely to ophthalmology.

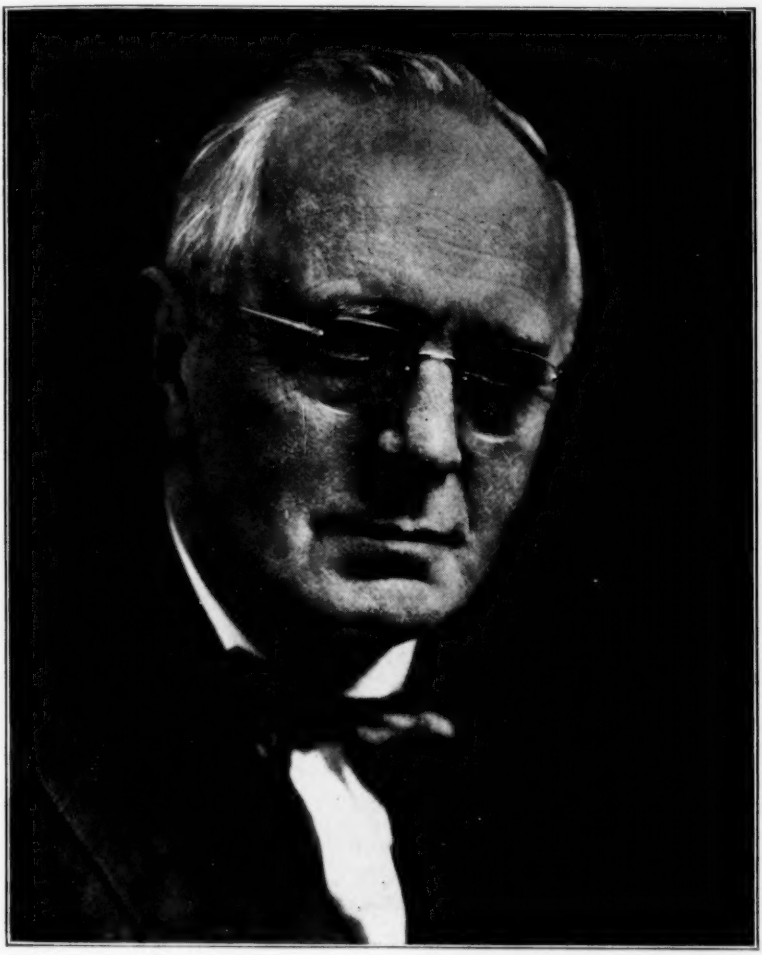
In 1930 Dr. Krug organized the ophthalmologic department of the Lenox Hill Hospital, which was ever near to his heart, giving to it of his time and means in fullest measure. He became associate professor of ophthalmology at the New York Post Graduate School of Medicine. He was a member of the American Academy of Ophthalmology and Otolaryngology, the American Ophthalmological Society, and a fellow of the New York Academy of Medicine. In World War I he served with the rank of major.

Dr. Krug's influence and reputation were in the main confined to those with whom he came in daily contact. He was a most excellent teacher and disciplinarian. In the course of time a succession of interns and clinical assistants had him to thank for the acquisition of a habit of paying scrupulous attention to details and for the example set by a chief who, free of envy, rejoiced more in the success of others than in his own.

His private practice was large and lucrative. Aside from remedies prescribed, his unfailing sympathy for his patients, rich and poor alike, enhanced the esteem in which the laity hold the medical profession. He has left the memory of a spotless name and of a life full of benevolences. To those of his colleagues who knew him intimately and enjoyed the hospitality of his home the sense of his loss is great and the goodness of his soul lasting.

Surviving are his wife, Louise Hoffman Krug; his sons, Ernest F. Krug and Dr. Joseph H. Krug, who is training to follow in the footsteps of his father as an ophthalmologist, and a sister, Miss Emilie L. Krug.

BERNARD SAMUELS, M.D.



ERNEST FREDERICK KRUG, M.D.
1877–1948

ELIAS SELINGER, M.D.
1898-1947

Dr. Elias Selinger, while at the peak of professional activity, died at El Paso, Texas, on Dec. 21, 1947, of coronary thrombosis, at the untimely age of 49. Just previous to his attack, he was busily directing the organization of the first postgraduate assembly sponsored by the Chicago Ophthalmological Society, of which he was then vice president, and was also zealously preparing for his duties as professor and chairman of the department of ophthalmology at the reorganized Chicago Medical School.

Dr. Selinger was born in Moravska-Ostrava, Czechoslovakia, on Aug. 23, 1898, the son of Joseph and Rosa Feuereisen Selinger. He came to this country at the age of 15 and graduated from the Northwestern University Medical School in 1924. After a general internship and a residency in the department of ophthalmology at Cook County Hospital, he continued his studies in Vienna. On his return, in 1926, he became interested in the teaching of ophthalmology, and served at Northwestern University from 1926 to 1929 and at Rush Medical College from 1929 until the dissolution of that venerable institution, in 1941, at which time he had achieved the rank of assistant professor. He received the certification of the American Board of Ophthalmology in 1931 and was a member of the American Academy of Ophthalmology and Otolaryngology, the Pan-American Association of Ophthalmology and the American Association for the Advancement of Science.

As an ophthalmic surgeon, Dr. Selinger was unusually painstaking in technic and postoperative care. His numerous contributions, usually given before the Chicago Ophthalmological Society, were based on personal research or extenive clinical observation and were always admirably presented. His recent textbook on "Office Treatment of the Eye" (reviewed in the ARCHIVES **39:** 118 [Jan.] 1948), though prepared in the turmoil of the war years, has his characteristic clarity and thoroughness.

At the time of his death he was attending ophthalmologist to Cook County, Michael Reese and Mount Sinai Hospitals. He is survived by a wife and daughter.

JAMES E. LEBENSOHN, M.D.

ELIAS SELINGER, M.D.
1898-1947

Correspondence

GLASS IRRIGATOR TIP IN ANTERIOR CHAMBER

To the Editor:—A case of ointment in the anterior chamber following a penetrating wound of the cornea was reported by Donald K. Binder, M.D., in the December 1947 issue of the ARCHIVES, page 830. I have seen several instances of ointment globules after operation for cataract prior to the introduction of closure of the section with corneoscleral sutures. It was not deemed advisable to open the anterior chamber in order to remove a globule because the antiseptic salve always absorbed.

A more unpleasant incident is brought to mind. Low grade inflammation was noted after a cataract extraction performed on my service in a large metropolitan hospital. Biomicroscopically, a glistening reflex was observed in an exudate near the superior limbus. Examination of the glass anterior chamber irrigators revealed one with a broken tip. After the wound was opened a small piece of glass was removed which exactly matched the defect in the irrigator tip. No complications followed.

When the incident described can occur in a modern and well organized operating room, I feel justified in condemning the use of irrigators with glass tips. The Bracken metal irrigator is safer and more effective. It can also serve as a substitue for an iris spatula.

JAMES W. SMITH, M.D., New York.

APPEAL FOR DONATIONS OF SLIT LAMPS AND OTHER INSTRUMENTS FOR CHINA

To the Editor:—For the past several years a nationwide campaign has been carried on in China to combat trachoma. This is largely supported by the National Blind Welfare Association, whose sister organization in the United States is the Institute for Chinese Blind, 156 Fifth Avenue, New York. The former secretary of the campaign organization, Dr. Pang Hsien Chen, writes that the need for instruments and supplies is most urgent. Six centers and twenty-one full time and part time clinics have been set up in Free China, but such equipment as slit lamps, ophthalmoscopes, operative instruments, tonometers, microscopes, etc., are lacking. Accordingly the Howe Libray of Ophthalmology is acting as a clearing house for the sending of available material to China. It is hoped that those ophthalmologists and others who have used slit lamps or other ophthalmologic equipment which they care to part with for this purpose will contact us and arrangements will be made, so long as funds for transportation become available, to send them to China. All communications should be addressed to Miss Jeanette Loessl, Howe Library of Ophthalmology, 243 Charles Street, Boston, Mass.

DAVID G. COGAN, M.D., Boston.

News and Notes

EDITED BY DR. W. L. BENEDICT

SOCIETY NEWS

Deutsche Ophthalmologische Gesellschaft Heidelberg.—This society held its fifty-fourth annual meeting in Heidelberg on Aug. 2 to 3, 1948. Prof. Dr. Engelking is secretary.

Ophthalmologic Society of the United Kingdom.—The annual congress of the Ophthalmological Society of the United Kingdom was held on April 8, 9 and 10, 1948, with A. J. Ballantyne, M.D., LL.D., president, presiding. The program consisted in a discussion of "Subjective Disorders of Vision" (excluding those due to local ocular diseases), which was opened by Prof. H. Cohen, Dr. Denis Williams and Mr. J. H. Doggat. The Bowman Lecture for 1948 was delivered by Prof. Marc Amsler, of Zurich, Switzerland, on "New Clinical Aspects of the Vegetative Eye."

The following papers were presented: "Ocular Palsies Due to Infection of the Nasal Sinuses," Dr. Helen Dimsdale and Mr. D. G. Phillips; "Preliminary Survey of Forty-Five Consecutive Cases of Congestive Glaucoma," Mr. J. P. F. Lloyd; "The Conjunctival Nevus and the Neurogenic Theory of Melanoma," Mr. Eugene Wolff; "The Venous Pressure in Glaucomatous Eyes," Dr. T. L. Thomassen; "Observations on Clinical Perimetry," Dr. G. I. Scott; "Two Cases of Special Interest:" (*a*) "A Subconjunctival Rupture of the Globe with Extensive Migration of Uveal Pigment," and (*b*) "An Aneurysmal Varix of the Retina," Mr. G. T. W. Cashell; "Papilledema Associated with Toxic Hydrocephalus," Mr. A. G. Cross; "Latent Nystagmus," Mr. T. Keith Lyle; "Classification of the Unassociated Dystrophies of the Fundus," Prof. Arnold Sorby; "Prognosis in Detachment of the Retina," Mr. C. Dee Shapland; "Mode of Development of the Vascular System of the Retina, with Observations on Its Significance for Certain Retinal Diseases," Dr. I. C. Michaelson; "Atropine in the Treatment of Glaucomatous Iridocyclitis," Prof. W. H. Melanowski. A film "Auto-Eversion of Upper Lid (left), Voluntary Unaided," was shown by Dr. W. C. Souter, and two pictorial demonstrations were made: "(*a*) Spasm of the Central Artery of Retina; (*b*) Entoptic View of Retinal Vascularization; (*c*) Hypophysial Tumor Causing Homonymous Hemianopsia; (*d*) Hypophysial Tumor Causing Paralysis of the Third Nerve, and (*e*) Depigmentation of Iris in Chronic Glaucoma," Dr. H. M. Traquair; "(*a*) Pressure Grafting in the Contracted Socket; (*b*) Epibulbar Dermoid Before and After Operation, and (*c*) Gummatous Ulceration of the Eyelids," Dr. J. Ellison.

Chengtu Eye, Ear, Nose and Throat Society.—The Chengtu Eye, Ear, Nose and Throat Society celebrated its tenth anniversary on Dec. 20, 1947. Dr. T. H. Lan, professor of biochemistry, West China Union University, the invited guest speaker, spoke on the biochemistry of blood in patients with malignant tumors. Dr. K. C. Lang and Dr. E. Chan

reviewed the history and progress of the society during the past decade. A photograph was taken to commemorate the eventful occasion, and a dinner concluded the session.

International Congress of Ophthalmology.—At a recent meeting of the International Council of Ophthalmology, held on April 7, 1948, in London, it was decided that the International Congress will meet in London in the third week of July 1950.

The following themes were chosen for the discussion: "Role of the Sympathetic Nervous System in Genesis of Vascular Hypertension and Its Effect on the Eye"; "Clinical and Social Aspects of Heredity in Ophthalmology." Madame Schiff-Wertheimer, Mr. Hedley Atkins and Prof. Wagner will open the discussion on the first theme; Dr. Schneyder, Miss Ida Mann and Professor Franceschetti, on the second.

The official languages will be English, French and Spanish.

The organizing committee consisted of Sir Stewart Duke-Elder, president; Mr. Frank W. Law, secretary, and Mr. E. F. King, Mr. T. Keith Lyle and Mr. A. Seymour Philps.

Centenary Congress of Section of Ophthalmology of Hungarian Medical Trade Union.—The Section of Ophthalmology of the Hungarian Medical Trade Union will celebrate its centenary congress in September 1948, at Budapest. The leading topics will be (1) Chemotherapeutics: Antibiotics in Ophthalmology, and (2) Modern Ophthalmic Instruments, Surgical and Optical. The president is Prof. Dr. Gusztav Horay, and the secretary, Dr. István Grósz, Nádor utca 32, Budapest V.

Ophthalmological Society of Australia.—The Ophthalmological Society of Australia, which is a branch of the British Medical Association, invited members of the Section on Ophthalmology of the Scientific Assembly of the American Medical Association to be represented at the annual meeting at Perth, Western Australia, Aug. 15 to 21, 1948.

Dr. Trygve Gunderson is secretary of the Section.

Association for Research in Ophthalmology.—The next meeting of the Association for Research in Ophthalmology will be held in Thorne Hall, Northwestern University Medical School campus, Chicago, June 21 and 22.

GENERAL NEWS

Home Study Courses Sponsored by American Academy of Ophthalmology and Otolaryngology.—The home study courses sponsored by the American Academy of Ophthalmology and Otolaryngology, in the basic sciences related to these two specialties, will be given again, beginning Sept. 1, 1948. Registrations must be completed before August 15. Detailed information may be secured from Dr. William L. Benedict, executive secretary, 100 First Avenue Building, Rochester, Minn.

Albrecht von Graefe's Archiv für Ophthalmologie.—This journal has resumed publication under the editorship of E. Engelking, W. Löhlein, O. Marchesani and K. Wessely. It is to be published by Springer-Verlag, Heidelberg and Berlin, and J. F. Bergmann, Munich.

The Albert D. Frost Memorial Library.—The Department of ophthalmology of the Ohio State University College of Medicine, Columbus, Ohio, announces the opening of the Albert D. Frost Memorial Library, following Mrs. Frost's gift of her husband's ophthalmologic library. Plans for a study room in connection with the library in the new medical center are being made. To maintain the library an endowment fund is needed, and an appeal is made to Dr. Frost's friends and others who might be interested in this worthy undertaking. Contributions of $5 to $25 are solicited; checks are to be made out to the Frost Memorial Library and sent to Arthur M. Cullen, M.D., University Hospital, the Ohio State University.

Delta Gamma Fraternity Fund.—The Delta Gamma Fraternity has a $1,000 annual fund from which smaller scholarship awards are available for preparation of persons intending to become (1) orthoptic technicians, (2) teachers of partially seeing children or (3) specialists for blind preschool children. Any one wishing to specialize in one of these fields may be eligible for assitance, the amount in each case to be determined by the particular need and costs involved. Candidates for training in one of these fields should apply for a scholarship to Mrs. Richard P. Miller, 39 West Jefferson Road, Pittsford, N. Y.

Candidates are selected with the advice of a professional committee: chairman, Le Grand H. Hardy, M.D., of the American Orthoptic Council; Mrs. Virginia Smith Boyce, administrative assistant, National Society for the Prevention of Blindness, Inc.; Miss Ruth E. Lewis, professor of social work, George Warren Brown School of Social Work, Washington University; Dr. Berthold Lowenfeld, director of educational research, American Foundation for the Blind; Miss Ruth B. McCoy, assistant director, New York State Commission for the Blind, and Lillian Ray Titcomb, M.D., president of executive committee, Nursery School for Visually Handicapped, Los Angeles.

Annual Examination for Orthoptic Technicians.—The annual examination of orthoptic technicians by the American Orthoptic Council will be conducted in September and October 1948.

The written examination will be nonassembled and will take place on Thursday, September 9, in certain assigned cities and offices and will be proctored by designated ophthalmologists.

The oral and practical examinations will be on Saturday, October 9, in Chicago, just preceding the meeting of the American Academy of Ophthalmology and Otolaryngology.

Application for examination will be received by the office of the secretary of the American Orthoptic Council, Dr. Frank D. Costenbader, 1605 Twenty-Second Street, N.W., Washington 8, D. C., and must be accompanied with the examination fee of $25.

Scholarships Established by the Lions Club of New York.—The Lions Club of New York has presented a check for $2,000 to the New York University College of Medicine to finance four scholarships for graduate training in ophthalmology.

The scholarships were awarded to Dr. Goodwin M. Breinin and Dr. Walter J. Maher, of New York; Dr. Edwin H. Kent, of Riverside, Conn., and Dr. Charles H. Toomey, of Lakewood, Ohio. They will study special phases of eye conservation at Bellevue Hospital under the direction of Dr. Daniel B. Kirby, chairman of the Department of Ophthalmology at the College of Medicine.

Argentine Congress of Ophthalmology.—The meeting of the Argentine Congress of Ophthalmology will be held in Mar del Plata from Dec. 13 to 18, 1948. The main subject will be "Tumors of the Visual Apparatus."

Medical Journals Needed Abroad.—Requests from libraries, medical societies and individual physicians in various devastated countries are being received almost daily. If physicians have extra copies of medical journals or copies for which they have no further use, it will be greatly appreciated if they will send them to the headquarters of the Association for distribution abroad.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Cornea and Sclera

ALLERGY TO ENDOGENOUS HORMONES AS A CAUSE OF KERATITIS ROSACEA. B. ZONDEK, J. LANDAU and Y. M. BROMBERG, Brit. J. OPHTH. **31:** 145 (March) 1947.

The authors supply the following summary:

Six patients (3 men and 3 women) with keratitis rosacea associated with rosacea faciei of long duration which had proved refractory to the usual methods of treatment were examined for hypersensitivity reactions to endogenous hormones. In all cases allergy to testosterone was demonstrated by positive cutaneous reactions to intracutaneous injection of the substance. Other hormones (estrone, estradiol [dihydrotheelin], progesterone, pregnandiol, corticosterone, insulin and gonadotropin) gave negative reactions. Desensitization treatment by a course of subcutaneous injections of testosterone in gradually increased doses and implantation of pellets containing 10 mg. of testosterone propionate produced satisfying results. Testosterone gave relief only when given in low, gradually increased doses; administration of large doses may cause a severe exacerbation. The finding of an allergic reaction to endogenous testosterone in a small number of cases of keratitis rosacea does not exclude the other possibilities.

W. ZENTMAYER.

Glaucoma

CYCLODIALYSIS: A FOLLOW-UP STUDY. H. S. SUGAR, Am. J. Ophth. **30:** 843 (July) 1947.

Sugar found that a relatively small number of successful cyclodialyses, after varying periods, became failures or relative failures. In Chicago, the recent trend has been away from cyclodialysis and toward iridencleisis. Cyclodialysis is more successful if the ciliary body is separated from the scleral spur along a third of the circumference of the angle, preferably by use of the Blaskovics technic.

W. S. REESE.

INVERSE CYCLODIALYSIS. R. N. SHAFFER, Am. J. Ophth. **30:** 860 (July) 1947.

Shaffer believes that so-called inverse cyclodialysis is safer and more successful, particularly if combined with injection of air. Gonioscopic control of the site of operation is essential. He reports 2 cases and gives statistics for 21 cases.

W. S. REESE.

THE RELATIVE VALUE OF SEVERAL DIAGNOSTIC TESTS FOR CHRONIC SIMPLE GLAUCOMA. S. BLOOMFIELD and L. KELLERMAN, Am. J. Ophth. **30:** 869 (July) 1947.

Bloomfield and Kellerman found the lability test the most dependable, the most convenient and the quickest of the diagnostic tests

for glaucoma. The water-drinking test followed closely in reliability. In decreasing order of dependability were the dark room test, the caffeine test and the response to mydriasis induced with paredrine hydrobromide ophthalmic (a 10 per cent solution of *p*-hydroxy-*a*-methylphenylethylamine hydrobromide in distilled water, made tear isotonic with 2 per cent boric acid and preserved with merthiolate, 1 : 50,000).

W. S. REESE.

Injuries

INTRAOCULAR FOREIGN BODIES IN SOLDIERS. H. C. WILDER, Am. J. Ophth. **31:** 57 (Jan.) 1948.

Wilder briefly discusses intraocular foreign bodies in soldiers from a study of 731 enucleated eyes at the Army Institute of Pathology. In 37 per cent they were magnetic, in 62 per cent nonmagnetic and in 1 per cent mixed. There were 5 eyes with sympathetic uveitis.

This study confirms previous observations that in military injuries nonmagnetic foreign bodies predominate, but many instances of successful extractions do not come to the pathologist's attention, and even in eyes that are enucleated nonmagnetic foreign bodies are often secondary to ferrous missiles, which have been extracted. The infections and reactions associated with the different types of foreign bodies ranged from staphylococcic infection, resulting in purulent panophthalmitis, to the mild inflammatory reaction associated with siderosis bulbi and intraocular hemorrhage without infection. Giant cells were particularly abundant around vegetable matter and eyelashes. Of particular interest were 5 cases of sympathetic uveitis, associated with retained nonmagnetic material in 4 cases and with a retained ferrous foreign body in 1. Phthisis bulbi was a common end result, and glaucoma was comparatively rare, as in any series of penetrating wounds.

W. ZENTMAYER.

INTRAOCULAR FOREIGN BODIES: AN ACCOUNT OF MILITARY CASES FROM THE BURMA-ASSAM FRONT. E. J. SOMERSET and K. SEN, Brit. J. Ophth. **32:** 13 (Jan.) 1948.

Operation was performed only in those cases in which two criteria were fulfilled: those in which there was a retained intraocular foreign body and those in which there was at least light perception with fair projection. A selected series of 29 cases are described. In 13 cases the foreign body could be seen with the ophthalmoscope. In 16 cases the ring method of localization was employed. Of the 6 anterior route operations, 5 were successful and 1 failed. In 23 cases the posterior route was used. In 17 cases, the foreign body was removed, and in 6 it failed to come out. The percentage of extractions was 75.8. With multiple foreign bodies, unless all came out, the operative result was recorded as a failure.

The author gives the following summary: A description of 29 cases of intraocular foreign bodies, mostly from Japanese grenades in the Assam-Burma Front is given. The low magnetic properties of the frag-

ments necessitated posterior route extraction in most cases. Ring localization proved accurate, and the Haab test was of little value, and was often misleading. The main causes of visual defect were vitreous hemorrhage and cataract.

W. ZENTMAYER.

Lacrimal Apparatus

LIPIODOL [IODIZED OIL U. S. P.] STUDIES OF CHRONIC DACRYOCYSTITIS. S. A. FOX, Am. J. Ophth. **30:** 878 (July) 1947.

Fox found that roentgenologic studies tended to confirm the belief that chronic dacryocystitis is an ascending infection from the nose or the paranasal sinuses, and that the dilatation of the sac is always forward and greater below the internal canthal ligament.

W. S. REESE.

Orbit, Eyeball and Accessory Sinuses

CONTRACTED SOCKET: THE SPLINT METHOD OF POST-OPERATIVE CONTROL. F. M. LYONS, Brit. J. Ophth. **31:** 703 (Nov.) 1947.

Briefly, the epidermal graft is applied to the prepared socket in the usual way, but on a special acrylic mold. At the first dressing (i. e., on the seventh day) the mold is connected to a rigid, but adjustable, splint. The fixed point is a frontal pin embedded in a plaster of paris head cap, and to this the mold is attached with stainless steel rods and universal clamps.

W. ZENTMAYER.

A PLEA FOR LATERAL ORBITOTOMY (KRÖNLEIN'S OPERATION). H. B. STALLARD, Brit. M. J. **1:** 408 (March 29) 1947.

Dandy, in 1943, stated that in 70 to 80 per cent of the cases of orbital neoplasm which cause unilateral proptosis both the orbit and the cranial cavity are involved, and he advocated the transfrontal route, as it is rarely possible before operation to be certain whether or not the tumor lies also within the cranial cavity. Harvey Jackson (*Proc. Roy. Soc. Med.* **38:** 587, 1945) stated "that by careful clinical examination, by judicious radiography and by suitable application of laboratory investigation, not only is the diagnosis of tumor to be reached but its ramifications regularly revealed and not infrequently its pathological nature surmised." Jackson concurred in Dandy's opinion that the transfrontal route is preferable in every way, as a more adequate exposure is obtainable and any extension intracranially can be approached. With this operation, there is less interference with ocular motility and there remains no visible and ugly scar, and it is not true that pulsating of the eye remains.

Stallard expresses the belief that lateral orbitotomy affords a more direct and more adequate exposure in cases of tumor limited to the orbit, and that good stereoscopic roentgenograms of the orbital walls and the optic foramens and canals are helpful in excluding extraorbital extension. There is no reason to fear an ugly scar.

He then describes a case of unilateral proptosis, due to a neurofibroma 30 by 23 by 16 mm. situated within the muscle cone and beneath the optic nerve. Transfrontal craniotomy failed to reveal the neoplasm, and six months later, after a temporary removal of the lateral portion of the orbit, the tumor was successfully removed. Vision recovered from perception of hand movements only to 6/6, and the field of vision improved by 35 degrees.

ARNOLD KNAPP.

Retina and Optic Nerve

A CASE OF PSEUDO-GLAUCOMA. J. A. MAGNUS, Brit. J. Ophth. **31**: 692 (Nov.) 1947.

A man aged 74 had vision of 6/9 + in the right eye and 6/9 part in the left eye. The right field showed a marked nasal step reaching fixation and a typical arcuate scotoma; the left field showed general peripheral depression. The field for red was disproportionately depressed. The intraocular tension was 19 mm. mercury and did not rise after instillation of homatropine. There was deep cupping of the optic disks. Roentgenogram showed calcification of the internal carotid artery.

W. ZENTMAYER.

SOLAR RETINITIS. E. ROSEN, Brit. J. Ophth. **32**: 23 (Jan.) 1948.

Rosen states that in the past year he has seen a great many cases, during routine examination, of small, easily overlooked macular holes. In all cases a history was elicited of "sun gazing" or a related act. The clinical history of 23 cases is tabulated, and fundus drawings of 21 cases are supplied.

The author has observed over 500 cases of small macular holes in "colored" troops, in none of which the patient was vasoneurotic and in every one of which there was a history of exposure to the direct action of the sun's rays.

W. ZENTMAYER.

ORIENTATIONS IN THE TREATMENT OF RETINAL THROMBOSIS. M. ESTEBAN, Arch. Soc. oftal. hispano-am. **4**: 106 (Jan.-Feb.) 1944.

This condition, which according to Schöbl occurs in 1 of 180,000 patients, and according to Dufour in 1 of every 5,000 patients, is usually considered from the ophthalmoscopic standpoint. Little, if anything, has been said about its treatment.

Three factors are concerned in its causation: alterations in the vascular endothelium, a blood status favoring rapid coagulation and slowing of the venous circulation. Treatment is directed to these factors by eliminating distant foci of infection; treating systemic infections with serum, vaccines and sulfonamide drugs; diminishing the clotting properties of the blood by use of sodium citrate, hirudin or heparin, and improving the local circulation by the use of derivatives, leaches and massage. A case is presented in which the treatment was used.

H. F. CARRASQUILLO.

Trachoma

PENICILLIN TREATMENT OF TRACHOMA. D. J. DARIUS, Am. J. Ophth. **28:** 1007 (Sept.) 1945.

Darius reports encouraging results in a series of 12 cases of trachoma treated with topical application of the sodium salt of penicillin in a solution of 500 units to each cubic centimeter of water. He reports 2 illustrative cases.

W. S. REESE.

TREATMENT OF TRACHOMA WITH ALBUCID [SULFACETIMIDE]. H. VALENCIA, Arch. Soc. de oftal. hispano-am. **3:** 432 (Nov.-Dec.) 1943.

The author's experience is in agreement with that of other observers as to the prompt disappearance of subjective symptoms and the rapid improvement of the corneal lesions of trachoma after the use of sulfacetimide, but he disagrees with authors who state that there is no action on the granulations and that the conjunctival swelling is diminished only at the beginning. Valencia has seen cases in which the granulations disappeared entirely in four days when sulfacetimide was used in large doses.

He draws the following conclusions: Sulfacetimide is efficacious in relieving the subjective symptoms and the corneal lesions of trachoma; it also acts on the specific conjunctival lesions when used locally; it is adjuvant in treatment, and the preparation is perfectly tolerated by the great majority of patients.

H. F. CARRASQUILLO.

Tumors

TUMORS OF THE EYE IN TURKEY. NURI FEHMI AYBERK, J. A. M. A. **135:** 373 (Oct. 11) 1947.

Before the Turkish Medical Society of Istanbul, Dr. Nuri Fehmi Ayberk reported his experience as ophthalmologist to the Haidar Pasha Model Hospital. He discussed at length 27 tumors of the eye encountered among the 70,000 ophthalmic patients he examined during the last ten years. There were 13 epitheliomas of the eyelids, 8 in men and 5 in women, between the ages of 40 and 70. Of the 27 tumors, 60 per cent were located at the inner angle of the eyelid. Dr. Ayberk attributed this location to age and occupation, advanced age causing relaxation of the orbicularis muscle and eversion of the lacrimal point, and the occupation of farming engaged in by the majority of these patients leading to chronic conjunctivitis, due to exposure of the eyes to strong sunlight and dust. These causes give rise to continuous lacrimation, causing irritation of the skin and obliging the patient to wipe the affected region often, thus facilitating the development of the tumor. Of the tumors of the conjunctiva, 1 was a papilloma arising on the upper eyelid; the other, resembling a melanosarcoma, proved to be a benign tumor originating at the lacrimal caruncle. Dr. Ayberk saw 7 carcinomas of the eyeball. There were 3 internal tumors of the eye, 1 primary sarcoma of the choroid and 2 gliomas. Of the tumors of the orbit, 1 was an osteoma and 1 a benign tumor of the optic nerve. In conclusion, Dr. Ayberk said he had not seen any tumors caused by parasites.

W. ZENTMAYER.

A Case of an Adenoma Arising in a Sweat Gland of the Upper Lid. F. Clifton and W. H. Gordon, Brit. J. Ophth. **31**: 697 (Nov.) 1947.

There was present a rather flattened, oval tumor with the long axis vertical and the lower pole overhanging the lid margin. The growth was encapsulated. The structure of the tumor resembled a tubulo-racemose gland. In places there was proliferation of the parenchymal cells as solid masses, between some of whose cells cell bridges could be made out. Many degenerative cysts, in all stages of development, were seen in the cell masses.

W. Zentmayer.

Uvea

Detachment of the Choroid and the Retina. G. H. Stine, Am. J. Ophth. **30**: 897 (July) 1947.

Stine, as an aid to correct diagnosis, describes the sign of ophthalmoscopic retroillumination or paraillumination, which is diminished or absent in cases of choroidal detachment and exaggerated in cases of serous retinal separation. He discusses anatomic and topographic relations of the vorticose veins and describes a method of orthographic projection to show precisely their action in localizing and limiting the size of choroidal detachments.

W. S. Reese.

Therapeutics

The Application of Penicillin in Ophthalmology. D. Berezinskaya, Vestnik oftal. **24**: 13, 1946.

Berezinskaya employed both Soviet and American-made penicillin. The Russian penicillin evidently has more bacteriostatic effect than the American penicillin, as the quantity required was much less than that of the American. In 1944 penicillin from Penicillium crustosum (Russian) was used in 20 cases in a military hospital, in a dose of 500 or 600 units three to four times a day. No local or general reaction was observed. In 1945 American penicillin was used in 14 cases, the dose being about 90,000 to 100,000 units a day. Penicillin gave excellent results in 13 cases with blepharitis and corneal ulcers. Of 11 cases of uveitis, penicillin therapy was effective in 4; of these, 2 were of tuberculous and 1 of rheumatic origin, and in 1 the cause was unknown. In the others the subjective symptoms were improved. In 1 case of sympathetic ophthalmia penicillin produced notable improvement in both eyes (the eye operated on for cataract, the exciting eye, was not removed, as vision in this eye was better than in the other) after two days of intramuscular injections. The treatment had to be repeated three times, after which vision was improved to 0.4 in one eye and to 0.8 in the other eye. In treatment of purulent infections of the lids and orbit local applications and intramuscular injections should be combined. In order to avoid the development of resistance to penicillin, the drug should be given intensively in the first stage of the infection.

O. Sitchevska.

Society Transactions

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COLLEGE OF PHYSICIANS, PHILADELPHIA, SECTION ON OPHTHALMOLOGY

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Factors Operative in Absorption from the Vitreous. DR. LUDWIG VON SALLMANN, New York.

In view of the difficulties in examining the exchange of fluids in the posterior segment of the eye by the orthodox method, a new approach was selected with the practical aspect of the problem in mind. The experiments were grouped (1) according to phenomena of diffusion in the vitreous and of its liquefaction; (2) in relation to the activity of the ciliary epithelium; (3) according to the influence of the basal metabolism, and (4) on the basis of clinical methods used to speed up absorption from the vitreous.

The dependence of the diffusion of hemoglobin on the structural integrity and the viscosity of the vitreous was studied by injecting pig's hemoglobin into fresh intact pig's vitreous. When the latter was exposed to a temperature of 37.5 C., the hemoglobin diffused throughout the vitreous within eighteen hours, whereas the spread was slow at refrigerator temperature. When hemoglobin was injected after autolysis of the vitreous had taken place on storage of the preparations for twenty-four hours at room temperatures, no difference in the diffusion of the hemoglobin was noticed at various temperatures. It was concluded that autolytic liquefaction, or lowering of the viscosity of the vitreous, had resulted in rapid diffusion in the eyes which had been exposed to 37.5 C. in the first series of experiments.

The effect of liquefaction on diffusion or spread and absorption of foreign matter was investigated in an extensive series of in vivo experiments on 24 rabbits. Partial liquefaction was induced by injecting various amounts of a purified and concentrated preparation of hyaluronidase made by Dr. Karl Meyer from bull testes; this mucolytic enzyme acts on the substrate of the vitreous. The enzyme dilutions were introduced together with suspensions of the animal's own red cells, which served as the test substance. Early spread of the red cells and moderately accelerated absorption were observed clinically and histologically in the eyes which had received more than 10 micrograms of the enzyme. When the dose exceeded 50 micrograms, the treatment elicited a conspicuous inflammatory response from the inner membranes of the eye. Even 2 of 5 eyes of the series receiving 50 micrograms reacted with pronounced signs of inflammation. The rest of that group and the eyes given injections of 10 or 25 micrograms exhibited early loosening of the clot of red cells and somewhat earlier absorption, without apparent toxic side action. It seemed that the partial liquefaction induced by the mucolytic enzyme caused the early spread of the red cells in the

vitreous, and this, in turn, may have facilitated the more rapid breakdown of the erythrocytes and their absorption. It is evident that at present the toxic action of a medium dose of the enzyme obviates its practical use.

The problem of active absorption possibly exercised by the ciliary epithelium was studied on the basis of R. Hoeber's work on the active transfer of a group of sulfonic dyestuffs through the wall of the proximal tubules of frog kidneys. Hoeber found that this transfer depended on the polar-nonpolar configuration of the dyestuffs, since the dyes with the sulfonic groups attached to one of the benzene or naphthalene rings were transferred through the wall, in contrast to the dyes in which each of the benzene or naphthalene rings carried a sulfonic radical. The present experiment on albino rabbits, albino rats and frogs with the use of various technics did not give evidence that the dye in the vitreous had penetrated the ciliary cells or had accumulated in intracellular vacuoles except in 1 of 60 eyes. Despite the negative results, further studies are warranted.

The influence of the basal metabolism on resorption was again studied on suspensions of the animal's own red cells injected intravitreally. Five litters of albino rats (34 animals) were divided into three groups so that litter mates were distributed among the three series. The first group, of 12 rats, were thyroidectomized by Dr. G. Smelser when the rats were 2 weeks old. The second group, of 12 animals, received daily subcutaneous injections of thyroxin, 1 mg. per hundred grams of body weight. After the injection of a small amount of the animal's own red cells, the density and extent of the cellular deposits in the vitreous were estimated by ophthalmoscopic examination and graded. The 68 eyes were examined three times a week, and the graded findings were tabulated so that the resorption could be expressed in curves when plotted against time. Thyroxin had no clear effect on the growth of the animals or on resorption of red cells from the vitreous, as compared with the results for a control series of 10 untreated normal animals. The thyroidectomized rats showed greatly retarded growth and, in general, flat resorption curves; that is, the red cells disappeared more slowly from the vitreous than in the 20 control eyes.

The last group of experiments was designed along the line of clinical methods used in the therapy of hemorrhages and other opacities of the vitreous. The effects of subcutaneous and subconjunctival injections of hypertonic solutions of sodium chloride, of iontophoresis and of artificial fever were examined. The animal's own red cells or a concentrated solution of insulin introduced into the vitreous was the test substance. Only the short bouts of fever induced daily for two weeks had in rats an indisputably beneficial effect on the rate of disappearance of the red cells from the vitreous (40 animals).

DISCUSSION

DR. IRVING H. LEOPOLD: The vitreous humor has been the subject of investigation by numerous men for many years and has involved a knowledge of special technics, of physical chemistry and of ophthalmology. Men such as Duke-Elder, Robertson, Hodgeson, Goedbled and Haurmann have worked on this problem for years. No one has contributed more to present knowledge of the vitreous than has Dr.

von Sallmann. It was a pleasure to hear him discourse on aspects of the vitreous, for on this subject he is certainly an authority.

The vitreous humor is a colloidal structure, and as a colloidal structure may exist as a sol or a gel. Sols are colloidal states in which the solid particles are dispersed in a fluid medium, and gels are colloidal states in which the solid particles, so to speak, join hands and form a framework in which the liquid medium is dispersed.

Most of the evidence—and much of the more recent evidence has been contributed by Dr. von Sallmann—indicates that the vitreous humor is a gel. It is not a very stable gel. The liquid and the solid phase are in unstable equilibrium, and this can be upset easily. When the vitreous gel is disturbed, the transparency of the vitreous is altered.

There are two ways, roughly, in which the transparency of the vitreous can be changed, with the production of opacities: first, by disturbance of the gel of the vitreous, and, second, by the influx of new materials, such as red cells, exudates and foreign bodies. An example of the first mechanism is the formations of senile opacities in the vitreous. These are thought to be due to the aging of the gel, which changes into the sol state. All gels tend to break down to the sol state as they age. Myopic opacities of the vitreous are thought to result from movement of the vitreous gel in an enlarged globe. Liquefaction of the vitreous gel by proteolytic enzymes may result in opacities.

The other group of opacities is due to the influx of new material, such as red cells, which Dr. von Sallmann has discussed. The problem of intraocular hemorrhage is a serious one. The treatment of hemorrhage in the interior chamber is still unsolved, but hemorrhage in the vitreous offers an even greater therapeutic problem. Treatment of hemorrhage in the vitreous is concerned not only with resorption of blood but with maintenance of the *status quo* of the vitreous gel, or its reconstruction if it is disturbed. Dr. von Sallmann has given us some ideas concerning the method of resorption of the cells, but, at the same time, he has suggested methods which disturb the structure of the vitreous body.

It is interesting to observe workers in other fields of medical science turning to the eye as an aid in solving their own problems. This has happened in the case of the diffusion factor. Dr. von Sallmann has reviewed their observations and has added his own in order to solve some ophthalmologic problems. Duran Reynals found that if vaccinia virus was injected into the testes of rabbits one could be more certain of "takes" than if the injections were made in other areas of the body. He could get a much severer infection with such injections. He then found that if an extract of the testes was injected into the skin prior to injections of vaccinia he could also facilitate systemic spread of the virus and increase the number of "takes." He thought that the testis extract facilitated the spread of the vaccinia by hydrolyzing the viscous mucoproteins in the connective tissues.

These diffusion factors were then found to hydrolyze hyaluronic acid. Karl Meyer and his co-workers isolated this mucopolysaccharide from the vitreous humor. Other investigators then turned to the vitreous, an excellent substrate of hyaluronic acid, as a place in which to compare and study the activity of various diffusion factors. They measured the ability of the various diffusion factors to reduce the viscosity of the

vitreous humor. Most such factors are thought to act through hydrolysis of hyaluronic acid. However, ascorbic acid is also known to reduce the viscosity of the vitreous. It does not work theoretically, as do the other diffusion factors; for that reason, many people do not consider it a factor in spread. Nevertheless, it can, when properly employed, reduce the viscosity of the vitreous humor, and it is quite possible that it may help in this manner in the resorption of hemorrhage in the vitreous. Vitamin C has been used in the past as therapy in intraocular hemorrhage. It was thought that it built up intercellular cement substance and reduced the capillary permeability, thus preventing further bleeding. It was not generally realized that it might reduce the viscosity of the vitreous and facilitate greater distribution of red cells and earlier resorption. It may not be as toxic as hyaluronidase and perhaps can be injected directly into the vitreous without producing the untoward effects of this substance.

The observations of the influence of thyroidectomy on resorption of blood are worthy of note. In 1936 Jeandelize and Drouet reported on cases of recurring hemorrhage in the vitreous. They reported that all their patients with this disorder had some degree of hyperthyroidism and expressed the belief that they could prevent recurrences by irradiating the pituitary body, thus producing lowered activity of the thyroid gland. It is interesting that the lower activity of the thyroid may work toward the prevention of hemorrhagic recurrences and, at the same time, retard the resorption of existing hemorrhages.

For years men have recommended fever therapy or shock therapy for resorption of hemorrhage in the vitreous, most of them without any well founded controlled experiments on which to base their choice of therapy. Dr. von Sallmann has given us controlled data to show the value of artificial fever.

There is some question about the circulation of fluid in the vitreous humor. As you know, the vitreous body is not thought to have any metabolism. It is supposed to be a product of the surrounding cells. It probably does not utilize oxygen or produce carbon dioxide. Regardless of this lack of necessity for metabolic interchange, there is some evidence of a flow of fluid through the vitreous. This has been demonstrated in rabbits, but not satisfactorily in man. The flow is thought to be from the ciliary body toward the optic nerve. Certainly, the diffusion of the red cells would not tend to substantiate the idea of such a direction of flow in the vitreous. The diffusion was in all directions of flow in the vitreous.

There is no doubt that papers such as Dr. von Sallmann's give one considerable information with which to further knowledge of the vitreous body and, at the same time, furnish concrete evidence on which to base future therapy of the disorders of the vitreous.

DR. FRANCIS HEED ADLER: I had not intended to discuss this paper, largely because of a feeling of inadequacy. I rise, as they say in parliamentary language, to a point of order, i. e., to object to the statement in Dr. von Sallman's paper in which he mentioned my name as having anything to do with furthering knowledge of the vitreous. Although long ago, in those halcyon days, I was interested in some of the physiologic problems of the vitreous, the work to which he refers is chiefly

a study in my laboratory carried out by Dr. Irving Leopold, and the full credit should be given to him.

Dr. von Sallmann's paper brings up many practical problems, and it is just such careful investigations as these which open the way to new therapeutic attacks on clinical problems.

From what has been said about hyaluronidase, it seems to me that the approach through its use is practically closed. The threshold of what is experimentally demonstrable in its effects lies too close to that which is also demonstrable in producing retinal damage. This situation is comparable to that of some anesthetics which perhaps are good anesthetics but for which the threshold of anesthesia is very close to that of death.

I should be interested in having Dr. von Sallmann comment further on thyroxin, particularly in relation to its water-binding properties, and the role of the hormone of the anterior lobe of the pituitary gland in the production of exophthalmos. I wonder whether he has taken into consideration the relation of thyroxin to the hormone of the anterior lobe of the pituitary in this respect, and whether he has anything further to say on this subject. I think that it is a lead which offers something.

Finally, I can confirm, as can all of us here, the clinical effects of fever therapy on the absorption of hemorrhage in the vitreous. That is certainly *fait accompli* and is one of the most reliable medical measures at present.

DR. LUDWIG VON SALLMANN, New York: I want to thank Dr. Leopold and Dr. Adler for their interesting discussions. I am sorry that I cannot offer anything on the significance of the thyroxin experiments to the function of the pituitary gland. I did not go into detail concerning the injections of thyroxin because the results were erratic. Sometimes a fairly significant increase in absorption of the red cells was obtained, but in other cases there were flat absorption curves.

DR. FRANCIS HEED ADLER: Why did you use thyroxin? What theoretic consideration led you to suspect that thyroxin might have some effect on the absorption of the hemorrhages in the vitreous?

DR. LUDWIG VON SALLMANN, New York: Hans Eppinger, in 1914, studied the effect of thyroid preparations on the water threshold and described the rapid absorption of tissue fluid in certain types of edema. These observations on the water threshold and the stimulation of the basal metabolism by thyroxin were the theoretic basis of my experiments.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, SECTION ON OPHTHALMOLOGY

Algernon B. Reese, M.D., *Secretary*

Fifty-First Annual Meeting, Chicago, Oct. 14-17, 1946

Beta Irradiation in Ophthalmology. DR. CHARLES E. ILIFF, Baltimore.

This paper, one of three read at a symposium on irradiation therapy in otolaryngology and ophthalmology, was published in a previous issue of the ARCHIVES (38: 415-441 [Oct.] 1947).

Tests of Heterophoria. Dr. Richard G. Scobee and Dr. Earl L. Green, St. Louis (by invitation).

The reproducibility or reliability of measurements of heterophoria when made by two examiners with the tangent screen–Maddox rod test was found in one sample of aviation cadets to be satisfactorily high. The coefficients of reliability were +0.95 for lateral heterophoria and +0.92 for vertical heterophoria at a testing distance of 20 feet (6 meters). In another sample, after removal of systematic effects due to the use of screening and to changing the color of the rod, the Maddox rod test both at 20 feet and at 13 inches (30 cm.) had a reliability coefficient of +0.97 for lateral heterophoria.

There is no significant difference in results obtained by two different examiners in administering the tangent screen–Maddox rod test.

Several tests for heterophoria were compared with the screen and parallax test which is considered the most accurate. At a testing distance of 20 feet, a high correlation was found with von Graefe's prism diplopia test and with the screen–Maddox rod test. At a testing distance of 13 inches, a high correlation was found with von Graefe's prism diplopia test, the Maddox wing test and the Thorington test. The correlation with the screen–Maddox rod test was somewhat lower in one experiment but high in another.

The effect of varying the testing conditions on the Maddox rod test was determined. The amount of illumination had no significant effect. It makes no difference whether the Maddox rod is placed over the dominant or the nondominant eye. At a testing distance of 20 feet a red Maddox rod uncovers more esophoria than does a white one but there is no difference between the effect of the red and that of the white rod at 13 inches. With screening the Maddox rod test uncovers more esophoria or less exophoria than without screening.

The Maddox rod test for all possible combinations of four alternative testing conditions was found to be highly correlated with the screen and parallax test.

The following conclusions may be drawn from the results:

(*a*) For heterophoria measurements may be performed with sufficient accuracy to discriminate between subjects.

(*b*) The Maddox rod test gives results sufficiently like those of the screen and parallax test to be used as a replacement for it for purposes of routine testing.

(*c*) It is recommended that the Maddox rod test be performed with a white rod, without screening and without regard to placing the rod before the dominant or the nondominant eye for purposes of routine testing.

DISCUSSION

Dr. John H. Dunnington, New York: The findings in this careful statistical survey of tests for heterophoria furnish much food for thought. The scrupulous care with which the tests were done makes their results all the more valuable. The authors have demonstrated that two careful examiners can reproduce more or less identical results with the various tests at one's command. They have compared the commonly employed methods and pointed out in a convincing manner the fluctuations one may expect to find in normal subjects with each

test. Their observations on the unimportance of (1) the amount of illumination and (2) the eye before which the Maddox rod is placed will do much to clarify the use of this test. Their observation that "with screening the Maddox rod test uncovers more esophoria or less exophoria than without screening" is in direct contradiction to the generally accepted idea, viz., that with the Maddox rod one usually finds a higher degree of esophoria or less exophoria than with the screen and parallax test. My only explanation for this discrepancy is that these tests were made on normal subjects, in whom all variations are reduced to a minimum. I believe that if similar tests were made on pathologic subjects these findings would be reversed. At least in my experience such has been the case. A similar objection can be raised to their conclusion: "The Maddox rod test gives results sufficiently like those of the screen and parallax test to be used as a replacement for it for purposes of routine testing." Since tests for heterophoria are dependent on suspension of fusion and the Maddox rod admittedly "does not completely abolish the fusion impulse," it seems to me that routine adoption of this test will necessarily lead to erroneous findings. These examinations were made on subjects with muscle measurements well within the normal limits. One should expect to find a minimal variation in this group, whereas if the test is used as the routine method much greater discrepancies will become manifest. The use of the screen with the Maddox rod was advocated by Dolman because during World War I this situation was found to exist. Several cases of high exophoria were detected with the screen and parallax test which had previously been missed with the Maddox rod test. In routine office practice the Maddox rod is particularly useful in the detection of small amounts of hyperphoria and is a valuable adjunct to our equipment for muscle testing, but I, for one, am not ready to adopt it routinely, to the exclusion of the screen and parallax test.

Such thorough analyses are so rare that I must congratulate the authors on their painstaking efforts and excellent presentation, from which all of us have learned a great deal. I only hope that similar investigations on a group of routine office patients will soon be forthcoming.

DR. GLEN G. GIBSON, Philadelphia: Dr. Scobee and Dr. Green are to be congratulated on making this scientific contribution to ophthalmology in the pressing circumstances of wartime military service. In my opinion, this is an important piece of work, because it focuses attention on a problem which is to become progressively more important, namely, the visual qualifications for aviation. Since the objective of all is to learn the truth about tests for heterophoria, it is probably fortunate that the military ophthalmologist is primarily concerned with the best of the population in the field of ocular muscle balance, while the clinical ophthalmologist is necessarily more interested in persons at the other extreme. Approaching the problem from these two extremes, one would anticipate greater differences of opinion than actually exist.

The authors have carefully studied an adequate group of subjects and have correctly concluded, with due regard to the limitations of their study, that two examiners can, with this selected group, make measurements with the screen–Maddox rod test which are repeatable and statistically reliable. I feel that in general this is quite true, as

day to day measurements of heterophoria probably do not vary much if the conditions of the test are repeated. Statements to the contrary in the literature refer, for the most part, to those with more pathologic cases than to patients with normal heterophoria. If screening adds only 0.3 to 0.4 Δ in the Maddox rod test to the reading without screening, I do not see the necessity of screening in the test, in which the clinical error is easily 1 Δ, as the authors prove. It is interesting and instructive that the authors demonstrate such an agreement in the screen-parallax test and the screen–Maddox rod test. In clinical work I have not been able to demonstrate routinely such a close parallelism, particularly with the very low and with the higher amounts of heterophoria.

One wonders whether the incentive to this work was a recognition by the authors that present technics and standards of testing were excluding good men from military service. I appreciate that it is not within the province of this paper to justify the standards of the Air Corps; but, as one who is unfamiliar with military standards, I find it difficult to understand why 5 Δ of exophoria is set as the dividing line between acceptable and nonacceptable men, and I should like to ask Dr. Scobee if he would care to state whether this is a fair end point for the discrimination between subjects.

The authors have permanently established that total darkness is not a requirement and that it is not necessary to place the rod before the nondominant eye. It is reassuring to have statistical confirmation of these aspects of the testing technic.

This paper manifests the necessity of more precise definitions, classifications and interpretations in the field of muscle balance, based on a better understanding of the reflex mechanisms involved. Most textbooks consider heterophoria as a subdivision of motor anomalies, when the problem is primarily a sensory one. What one is measuring is the motor compensation for sensory conditions. Most cases of heterophoria are instances of imperfection, but they are not anomalies. There is need of more precise definition of the difference between normal heterophoria and clinically significant heterophoria.

Fusion does not perform a motor task, as the authors state. Fusion is a purely sensory process, which occurs after the fusion-vergence reflexes have produced the motor adjustment necessary to bring the eyes from the disassociated to the associated position. The fact that this is an insignificant task in the great majority of cases should be appreciated in setting standards for this subject. I feel that many of the points the authors have made will stand the test of time and will prove of permanent value, and it is hoped that these investigators will make a similar survey on patients with clinically significant heterophoria in the near future.

I wish to express to them my sincere thanks for this instructive paper.

Dr. William F. Bonner, Temple, Texas: In a paper presented before the Philadelphia College of Physicians in December 1923, on "The Relation of the Weakness of the Extraocular Muscles and Depth Perception," based on two groups of patients, one with premature presbyopia and one with normal near vision, I said:

> In patients with premature presbyopia: (1) Exophoria combined with good depth perception; (2) esophoria combined with poor depth perception; (3) orthophoria combined with good depth perception, except in cases of high latent hyperopia, in which the depth perception was poor.

In patients with normal near vision: As a rule, exophoria combined with good depth perception; low degrees of esophoria combined with good depth perception; high degrees of esophoria combined with poor depth perception, and right hyperphoria combined with poor depth perception, but hyperphoria and depth perception were improved by correcting lens and prism.

DR. RICHARD G. SCOBEE, St. Louis: The problem of repeating the test on "abnormal subjects" is, of course, obvious. We have already tested approximately 100 patients in clincal practice, and we are beginning work on a separate group who are definitely abnormal, to see whether the test is reproducable there, as it obviously seems to be with normal subjects. Certainly such a step is indicated.

In answer to Dr. Dunnington: It was not intended that the screen–Maddox rod test should in any sense replace the screen and parallax test. The idea was to show that if one takes 10 patients and ranks them in order of the decreasing degree of heterophoria, from 1 to 10, or vice versa, and if one tests these patients with the screen and parallax tests, 9, roughly speaking, will rank in the same order of degree of heterophoria, and the parallax test will uncover more exophoria than will the Maddox rod test. Certainly, I should be the last to detract from the value of the screen and parallax test, which I fully agree with Dr. Dunnington is the most accurate test of heterophoria available. The Maddox rod test was not recommended as a substitute, but was mentioned simply for comparison. If some one sends a patient to you and says, "This patient has this amount of heterophoria," and you say, "Oh, no, he has this much," you are both sincere and you are both right, but you are using different tests of heterophoria.

Dr. Gibson made the same point.

The question was raised as to how this work began. We wanted to know why the original limits were selected in the Army Air Forces, as they were, of 5 D. of exophoria and 10 D. of esophoria for class 1 pilots. There seemed to be no good reason, and certainly I cannot suggest any answer. They must have been pulled out of a hat. We hope by a study of the normal to be able to predict what might be considered abnormal.

Effect of Visual Training on Existing Myopia. DR. H. ROMMEL HILDRETH, DR. WILLIAM H. MEINBERG, DR. BENJAMIN MILDER, DR. LAWRENCE T. POST and DR. T. E. SANDERS, St. Louis.

In an attempt to improve the visual acuity, 54 selected patients with myopia were given a course of visual training, which was preceded and followed by a complete ophthalmologic examination.

The technic of training, which was based on standard accepted procedures in this field, was given by an optometrist, while the pretraining and post-training examinations were made by a group of ophthalmologists.

Thirty, or 55.5 per cent, of the subjects showed no change in their acuity, while 12, or 22.2 per cent, showed definite improvement, the best results being obtained in the patients with small amounts of myopia. Twelve patients, that is, 22.2 per cent, showed a change so slight as to

be excluded in the results. The change in the group with improved vision averaged 27 per cent. Eleven of the 12 patients who showed improvement were rechecked at intervals of fifteen to twenty-three months after completion of training. Five of these retained their improvement, while only 1 had less than the pretraining acuity.

There was no change in the refractive error or significant alteration in the neuromuscular mechanism of the eyes. It was thought that the improvement occurred because of improved reception due to stimulation of the visual effort, as the best results were obtained in patients with a pretraining acuity less than would be expected from their refractive error, 73 per cent of this type showing improvement.

Visual training has a definite, but limited, value with some myopic patients, particularly those whose vision does not correspond with their known myopia. The effectiveness of visual training must be increased if it is to be generally applicable. Visual training merits further study from the ophthalmologist, particularly in relation to progress in treatment of myopia.

DISCUSSION

Dr. Alan C. Woods, Baltimore: Dr. Hildreth, Dr. Post and their collaborators deserve the thanks of this society for their meticulous investigation of this plaguing and perplexing problem of the value of ocular exercises in the treatment of myopia. None but the five who have labored on such an investigation can appreciate the endless and tiresome labor entailed. I congratulate Dr. Hildreth, Dr. Post and their associates, not only on the thoroughnes and conscientiousness with which this study has been set up and executed but also on the clarity of their presentation.

My only experience with this subject has already been set forth in my report from the Wilmer Institute on a closely similar investigation made by Dr. Frank B. Walsh and associates on the staff of that institution (*Am. J. Ophth.* **29:** 28, 1946).

The investigations reported here differ from the Baltimore report only in several minor respects: (1) High degrees of myopia or astigmatism were excluded; (2) a full correction to 20/20 vision was required in each eye; (3) normal muscle balance was required. In our series we took all grades of myopia up to 10 D. A full correction to 20/20 vision was not required, although this was the visual level of 81 of the subjects. Eighteen had vision of 20/30+ in each eye; 2 had from 20/50 to 20/30, and 2 had unilateral amblyopia with vision of 20/20+ in the second eye. We did not require a normal muscle balance or investigate the question of binocular vision, our investigation being conducted on the basis of improvement in the individual eye rather than in binocular vision. We tested both the pretraining and the posttraining visual acuity on four charts: the Snellen letter, the Snellen number and the Snellen E chart, and a modified Landolt broken ring chart, taking as the final visual acuity the mean for these four tests. Further, in the St. Louis investigations the visual training was conducted under the direction of Dr. Hildreth and Dr. Post, while in the Baltimore investigation it was conducted independently by a group of optometrists and experimental psychologists, under the direction of Dr. A. M. Skeffington and Dr. S. Renshaw. The two courses of visual training,

however, appear to be closely similar, if not identical. Despite these differences, which I believe are unimportant, the results of the two investigations are startlingly similar.

Drs. Hildreth and Post found that in 42 of their 54 patients, or 77.8 per cent, the final vision was unaffected by the visual training, while in 12 patients, or 22.2 per cent, there was definite improvement. We found no significant change in the visual acuity in 73 of our 103 patients, or in 71 per cent, while in 30, or 29 per cent, there was definite improvement. In both experiments the average degree of improvement was exactly the same—27 points in the visual efficiency scale.

On only 9 of our patients showing this improvement were we able to obtain later examinations to see whether the improvement was maintained. In 2 of these patients the improvement had been fully maintained; in 4 it had been partially maintained, while in 3 nearly all had been lost. Drs. Hildreth and Post found the improvement maintained in 50 per cent of their patients showing improvement. The difference in these figures is of no statistical significance. Both the Baltimore and the St. Louis group found that the visual training had produced no change whatever in the underlying refractive error. We concluded that the improvement noted in 29 per cent of our patients was due to teaching the patient to interpret his blurred retinal images more carefully. Dr. Hildreth and Dr. Post concluded that the improvement occurred at the cerebral, rather than at the retinal, level—a happy phrase!

Thus, the results of these two studies are practically identical, and the conclusions drawn from them by two entirely separate groups of investigators are the same, with one exception. Drs. Hildreth and Post state: "We must conclude that visual training has a definite, but limited, value with some myopic patients." We concluded, "The visual training used on these patients was of no value in the treatment of myopia." Thus, we have the paradox of two groups of investigators pursuing the same investigation, reaching results which are identical and yet drawing radically different conclusions as to the value of the visual training. I should like to defend my point of view.

The conclusion of Drs. Hildreth, Post and their associates is clearly based on two facts, namely, (*a*) that 22.2 per cent of their patients showed improvement of 27 points in the percentage visual efficiency, and (*b*) that 50 per cent of these patients permanently retained the improvement. The question before us is whether these facts are significant.

In Major Pincus' paper on correlation of the unaided visual acuities with refractive errors, he specifically noted that in patients with identical refractive errors, i. e., +1.50 D. sph.—1.00 D. cyl., axis 180, the vision may vary from 20/100 to 20/300, or from a percentage visual efficiency of 48.9 to one of 8.2, a range of 40.7 points. In our series, we found that patients with exactly the same refractive error likewise varied up to 42 points in the percentage visual efficiency, the mean variation being 29 points. We found, further, that when the same subject was tested with the four different charts at the same examination he might show an even higher variation, swings of over 50 points in the visual efficiency being observed on different charts. In evaluating any

beneficial results, one should, therefore, first determine whether in the pretraining determination of visual acuity the patients who showed the definite improvement had higher, average or lower visual acuities than would be expected with their refractive errors. If the pretraining visual acuity was higher than would be normally expected, any improvement obtained would be the more significant; if lower than would be normally expected, the improvement would lose its significance.

Analysis of the 30 cases in the Baltimore series and 11 of the cases in the St. Louis series in which vision improved shows that the pretraining visual acuity was the average to be expected with the refractive errors. Thus, in the Baltimore group, 7 subjects had higher than expected uncorrected vision, 16 had about the expected vison and 7 had lower than the expected vision. In the St. Louis group, 4 had higher than expected vision, 4 had the average expected vision and 3 had lower than the expected vision. If 40+ points is accepted as the normal variation in vision with the average refractive error, a swing of 20+ points in the average subject is without significance. The average improvement noted in the groups benefited by the visual training was 27 points for each series.

In my report, I called attention to the fact that the experiment was uncontrolled. This is also true of the St. Louis investigation. If the results of an experiment are negative, the absence of controls is unimportant; but when positive, these results must be interpreted against the behavior of a control group. We found it impossible to assemble a proper group of controls, and I have no doubt Dr. Hildreth and Dr. Post encountered the same difficulty. A proper control in this experiment would be an equal number of intelligent myopic patients with refractive errors and vision comparable to those in the training group. This control group should have their uncorrected vision determined, and they should be advised that patients with myopia of the same degree may have different levels of vision on the Snellen chart, depending on the care with which they interpret their own blurred retinal images. They should then be dismissed to follow their usual lives, with their vision uncorrected and with no visual training save that which they gave themselves by being more painstaking in their visual habits. At the end of training, given the test group of myopic patients, the control group should be reassembled and their visual acuities again determined, to ascertain what change, if any, had been accomplished by the simple explanation of the situation and the advice to be careful in their visual interpretations. What the results would be in such a control group, one can only surmise. I should not be surprised if 25 per cent showed an average improvement of 27 points in their visual efficiency.

In the St. Louis investigation, Dr. Hildreth, Dr. Post and their associates found that 50 per cent of patients showing improvement maintained their gain, while 50 per cent reverted to their former sloppy visual habits and lost what they had gained. In other words, 5 of 54 patients, roughly 10 per cent, showed final definite improvement. Our figures on this point are not so accurate, since we were able to recapture only 9 of our 30 patients showing improvement for the later, final, examination. However, accepting the higher, and more convincing, figures of the St. Louis group, one may well ask: Is this gain of 27

points in the visual efficiency, attained in only 10 per cent of the subjects, worth the time and the effort? This improvement corresponds to a gain in visual acuity of from 20/200 to 20/100, or, in the lower brackets, from 20/80 to 20/40. It is noteworthy that even among the trainees with the lower degrees of myopia, only 2 gained the 20/20 level, improving from 20/30.

To my mind, these meager results do not justify the expenditure of the time, the effort or the expense normally entailed by such visual training. My admiration for Dr. Post as a chairman, an administrator, an investigator and a friend is unbounded. In this instance, however, I am unconvinced. On the evidence so far advanced, I do not believe that visual training has a definite, or even limited, value in the practical treatment of myopia. It may have, in some cases, a psychologic value in reconciling the myopic person to his handicap. All in all, however, I believe that visual training should regretfully be consigned to the limbo of well intended, but fruitless, experiments.

DR. KENNETH C. SWAN, Portland, Ore.: We are indebted to the speakers and their colleagues for providing us with statistical data for the discussion of a well publicized controversial problem. We can best attempt to pay our debt to these men for their tedious and intensive work by properly interpreting it to the public, to optometrists and to our colleagues.

Both authors stress that their studies were limited to visual acuity, but the training procedures described in the published report would be beneficial to patients with anomalies of convergence and accommodation. Dr. Post included no cases of such anomalies in his report, but they commonly accompany uncorrected myopia. This fact must be taken into consideration in discussing with patients visual training for myopia. For example, a young, competent attorney stated that he was receiving great benefit from visual training administered by an optometrist. It was expensive, but he was going to continue because until he had visual training he had been unable to read without glasses. Spurred by the improvement in his reading ability without glasses, this man falsely hoped to discard his glasses for distant vision. I say "falsely" because an analysis of his previous symptoms reveals that he was able to read with one eye at a time before training, but that when he attempted to use both eyes the print ran together. He had other symptoms typical of convergence insufficiency. So, in discussing visual training with one's patients, one should not be satisfied to say that visual training does not alter the refractive error. One should add that in a few cases increased visual performance may result from improved coordination of the eyes and better interpretation.

I should like to call attention to another class of patients who are not uncommon, and who fall into the hands of quacks unless properly directed to psychiatrists. These patients characteristically profess to have received great improvement in their visual acuity from training, but an objective change cannot be demonstrated. Rationalization is of no avail, for this is no simple matter of vanity concerning glasses. For example, the intelligent daughter of a university professor claimed to have obtained great improvement from expensive training received from an optometrist, but objectively I was unable to demonstrate improvement in her vision. This patient was of a schizoid personality.

This type of person expresses subconscious thoughts in a distorted form. When this girl wore her myopic correction, she saw sharply and was aware of the faces of other people. They appeared to be staring at her; consequently, with her glasses she was awkward and self conscious. She was in a hard, sharp world of critics. Without glasses people's faces seemed blurred, but the girl was less conscious of them as individuals and felt at ease. It was a soft, blurred and kindly world. Unconsciously, this girl had accepted visual training as a means of staying in this friendly world. Rationalization and attempts to force this girl to accept the fact that she was getting no benefit from training might have done her harm. The oculist must direct patients to psychiatrists rather than drive them into the hands of quacks.

Dr. Post and Dr. Woods brought out the point that the improvement in visual acuity is on a mental level. The ophthalmologist uses two simple slides to demonstrate to his myopic patients the mechanism by which improvement in visual acuity occurs for a time after they discontinue wearing glasses. Experience, rather than visual training, is the deciding factor. On the first slide, any child can recognize that these simple block letters are T-H-E. The second slide shows only the shadows cast by the letters. The actual lines of the letters are missing, but the average adult mentally fills in the defects from experience and interprets the letters as three dimensional. On the other hand, the average child who has had no experience with three dimensional letters, has difficulty in reading the word. In other words, one demonstrates to the patient that with or without training he will, by experience, learn to interpret and to fill in incomplete or blurred images when he discontinues his myopic correction.

DR. S. JUDD BEACH, Portland, Maine: It is high time that the so-called cures for myopia by unorthodox methods be evaluated. We are indebted to Dr. Post and, previously, to Dr. Woods and their associates for these thorough investigations. It is generally believed that the gain is in the improved interpretation of blurred images, a position previously taken by Duke-Elder.

Though this is doubtless true of most cases studied, in last year's *Transactions* I described a different sort of case. A patient trained to squeeze his lids together was able to improve his vision during that act from about 4/200 to approximately 20/30. One keen observer thought he actually shortened the globe; another expressed the belief that the effect was simply the pinhole phenomenon.

I have since found in my office practice that this is a knack possessed only by part of the myopic patients. It is not purely the result of pinhole vision as some, while looking through a pinhole, can still further improve their visual acuity by squeezing the lids together. An 18 year old youth, whose relaxed vision was 2/60, with a 1 mm. pinhole saw 6/60 in one eye and 6/30 in the other, but with the same pinhole plus squeezing he saw 6/20 in the eye with 6/60 vision and 6/20—1 in the other eye.

It will be recalled that Bates and his followers, in this country, and Simpson, in England, teach that by action of the extrinsic muscles they produce genuine shortening of the eyeball. Such a result can be conceived as effected by the orbicularis muscle. It was certainly not a pinhole phenomenon in the cases I have described. Admitting that the

improvement is mostly at the cerebral level, one should not wholly close one's mind to other possibilities.

DR. WILLIAM H. CRISP, Denver: The patient's judgment, experience and general cerebral capacity are important in visual training. Sometimes, of course, peculiar ways of thinking about things may account for the general objection to wearing myopic glasses. I had a patient several years ago who complained about the glasses that I had prescribed for her. She said, "Doctor, I cannot possibly wear these glasses because when I sit at a table I see every pore of the person in front of me." The anatomic basis that has been reported on by Dr. Woods and by Dr. Post and his colleagues seems to be the determining factor in a myopic patient's giving credence to the claims made by persons who charge $175 for improving his vision.

If the patient's anatomic condition remains the same, I cannot see the ultimate advantage of these so-called exercises. A patient stated that he paid an optometrist $175 to get him into the Army because he was not able to get in on the first test. His anatomic abnormality was just as bad as it was before; and, generally speaking, he had gained nothing toward getting into the Army.

Almost any fairly intelligent patient would gain somewhat by experience in interpreting what is seen on the test card. It is the experience of most ophthalmologists that an illiterate patient who says he does not know the letters but knows the numbers has difficulty in recognizing numbers, a difficulty which, obviously, is due to lack of familiarity with the particular shapes of the numbers on the chart, whereas he can recognize the numbers more easily if they are shaped like those with which he is familiar.

I have had a number of patients who, at the first examination and the first fitting for a myopic correction, could read down only to a certain line on the chart, and who six months or a year later could read a line or two or three lines farther down the chart. Surely, some value must be given to experience of things seen which could not be seen before the correction was worn, and judgment comes into that result, just as it came into the responses of the patients reported on by the investigators today.

DR. BENJAMIN MILDER, St. Louis: I should like to thank Drs. Woods, Swan, Beach and Crisp for their interesting and thought-provoking comments on this highly controversial, and scarcely yet touched, field.

I believe that it is possible to see in Dr. Woods's comments a significant relation of the various points that he has discussed. They make it seem that, having tried this new method, and not having attained an extremely significant and highly remarkable positive result, one should therefore reject it and return, shall we say, to the ostrich attitude regarding this problem which has been prevalent in ophthalmology prior to this time.

A careful consideration of the paper read by Dr. Post would indicate that our approach has been a carefully limited and highly conservative one. Such an approach explains why we limited so carefully the patients we studied in this investigation. We felt that readings for people on the fringe, at the edges of the distribution curve, might throw off our

results, so that we might get an abnormal improvement or abnormally poor results.

With regard to the question of binocular vision and the evaluation of binocular vision, it should be pointed out, although that part of the paper was not read, that all the training procedures utilized by the persons performing these tests were procedures in binocularity, and therefore it is only logical that, since visual acuity as such in the average person is a binocular function and the tests were binocular tests, one should evaluate the vision of the two eyes together.

I should like, further, to read a sentence that Dr. Woods quoted from our paper, Dr. Woods feels that the 25 per cent or so of patients showing definite improvement which was obtained both in his study and in ours has no significance in the treatment of myopia. He quoted from our paper: "We must conclude that visual training has a definite, but limited, value with some myopic patients, preponderantly those whose vision does not correspond with their known myopia." I should like to read the sentence which follows: "Considering the time and effort expended by the patient, the results, both in the percentage of patients improved and in the actual amount of improvement per patient, must be increased if the procedure is to be generally applicable to the routine management of myopia."

It is entirely possible that future studies will demonstrate that there is no applicability or clinical significance in this type of training in the treatment of myopia. However, when one sees oil on the ground, one usually digs for oil there, and not at some barren part of the ground where one does not suspect it to be.

Dr. Swan mentioned the relation of muscle anomalies and malcorrected myopia. His point is well taken, and I believe that this would be the next logical point of departure in the investigation of the effect of visual training on myopia. I believe there is nothing unusual, or even surprising, about the success that we achieved in this investigation. All are acquainted with the fact that in amblyopia ex anopsia, which is an instance of a visual organ not being utilized to its fullest capacity, one sometimes sees with even simpler methods a much more remarkable improvement in the visual acuity.

Preoperative Roentgenographic Reconstruction of the Orbit.

LIEUTENANT COLONEL E. L. SHIFLETT, Medical Corps, Army of the United States.

My colleagues and I have many patients with badly comminuted fractures of the orbital and facial bones who are undergoing surgical reconstruction of the orbit. Success in this field depends to a large extent on restoration of the orbit by replacement of fragments, removal of fragments or substitution of tissue or material for lost bone tissue. The correct total diagnosis of bony deformities is shared by the roentgenologist and often is dependent on him almost entirely.

Stereoscopic posteroanterior roentgenograms, with the patient in the chin-nose position, the nose and chin resting heavily on the film, and stereoscopic lateral views, in the injured side down, are made. The films are processed in the usual way, and all are studied stereoscopically and pseudoscopically. Under stereoscopic vision the normal and abnormal bone structure is marked on one of the pair of films with an indel-

ible pencil, the normal orbit being used as a guide. After all fractures are identified and defects in the orbit created by the fractures noted and traced on the film, the film is removed and placed on an ordinary viewing box. The preliminary tracing just described is traced on the right side of the film as the patient faces the stereoscope, provided the observer is right handed; the left side of the film is traced if the observer is left handed. After this tracing is placed on the ordinary view box, which lies in the horizontal position, a piece of clear transparent paper is placed over the film and the tracing is recopied on the paper.

Our studies of reconstruction subjected to surgical control led us to certain general conclusions, with which the surgeon may or may not agree. The cephalocaudad position of the globe is important in that it might be used as an indicator for conservative or radical elevation of the orbital floor. It would certainly indicate that the same elevation is not applicable to both the high and the low-lying globe. Restoration of lost tissue, without a preoperative method of measurement, is usually too conservative. The most evident deformity is not always the chief offender as the cause of an unsightly prosthesis or malposition of the eye. Correction of the lesser of several injuries will often give a good cosmetic result which could not be obtained by correcting only the most evident injury, and a decision as to which defect to correct cannot always be determined by inspection and palpation alone. Unrecognized expansile intraorbital fractures are frequently the cause of the sunken, and otherwise misplaced, prosthesis. The size of the defect in the orbital floor is not necessarily compatible with global malposition or a displaced prosthesis, as the prolapse of orbital tissue through a small defect will cause malposition of both. It is possible that the position of the opaque prosthetic ball has some influence on the position of the artificial eye. Difficulty in attaching the superior paddle of the implant eye can be anticipated and prepared for by the simple procedure of orbital reconstruction. This procedure will aid the surgeon to measure and to shape with reasonable accuracy the tissue or material necessary for restoration before the patient is brought to the operating room.

Practical applications of this method were discovered and utilized in the surgical restoration of the wounded orbit. We believe that persistent study and consistent use of this method, or some other suitable method or preoperative reconstruction, will aid the surgeon and benefit the patient. We offer this method only as a practical aid to the surgeon, believing that it has a high degree of clinical and surgical accuracy, but do not offer it as representing in any way a physical or anatomic formula of complete accuracy. The method was developed by mutual assistance and cooperation of the ophthalmologic and roentgenologic services, and neither service could have developed it independently.

DISCUSSION

LIEUT. COL. GILBERT C. STRUBLE, M.C., A.U.S.: This method of orbital tracings and preoperative reconstruction of the orbits devised by Colonel Shiflett has proved to be of extreme value in the work on orbital reconstruction at our hospital. Since its development, the procedure has been routine in all our cases prior to operation. I have chosen the term "orbital blow-outs" as being most descriptive of

many of these injuries. They occur as a result of the terrific impact produced by the modern high velocity missiles. Ophthalmologists are also seeing cases of a somewhat similar nature as a result of accidents incurred in civilian life. Enophthalmos may be so pronounced that at times only the up-turned cornea of the globe is visible. In the majority of these cases, however, the eye has been destroyed and has been removed before the patient reaches us.

Correction in these cases requires elevation and restoration of the orbital contents to their normal position and filling of the defect with either preserved cartilage or some inert material, such as tantalum mesh, as devised and developed by Dr. A. D. Ruedemann. With tracings available, the required thickness of the cartilage, tantalum or other type of packing utilized can be estimated to an accuracy of 1 or 2 mm.

In many instances ordinary examination, including routine roentgenographic procedures and palpation of the orbital margins, has proved misleading in our attempt to evaluate the extent of injury present. Such an accurate analysis of the true extent of injury is important, since certain patients on whom a cosmetic operation might result in fatality may be eliminated from surgical consideration entirely. This was well demonstrated in 2 cases which at first appeared well suited to the usual surgical procedure. Preoperative orbital tracings in both cases, however, indicated a severe loss of bone of the posterior orbital walls, with the probability of herniation of brain tissue underneath the conjunctiva, a condition which was later confirmed by the neurosurgeon in both instances. [A case of this nature was presented.]

A surgical approach through the conjunctiva is advocated. It leaves no external scar and in our hands has proved more satisfactory than the incision through the skin. Dr. A. D. Ruedemann, the civilian ophthalmic consultant to Crile General Hospital, devised this technic. He spent hours with us each week and gave us help and suggestions in the development of these procedures.

[Three case reports were then presented, with preoperative and postoperative photographs. The orbital tracings in these cases made before and after operation were reviewed.] In 1 case a severe fracture involved the orbital floor, the entire ethmoid capsule and the cribriform plate, resulting in complete loss of sense of smell bilaterally. At the time of the original orbital reconstruction the depressed area in the posterior superior aspect of the ethmoid labyrinth was not corrected because of the possibility of opening up the old fracture lines in this area, which extended into the anterior fossa of the skull. In spite of this precaution, some drainage of cerebrospinal fluid from the orbital incision developed, together with rather severe headaches for the first three or four days after operation. After administration of penicillin and sufadiazine in adequate doses, the headaches disappeared and the operative orbital approach healed without further complication, with complete cessation of drainage of cerebrospinal fluid. The ultimate cosmetic correction in this case was not satisfactory because of the incomplete correction aforementioned. This resulted in some residual depression in the superior nasal aspect of the upper lid. This mild deformity was subsequently corrected by a second stage proce-

dure, which consisted in placing a curved acrylic contour stent between the orbital fascia and the orbicularis muscle, as advocated by Sugar and Forestner (*Am. J. Ophth.* **29:** 993, 1946). A short 16 mm. "kodachrome" moving picture was shown, demonstrating the use of orbital tracings and the operative procedure.

Colonel Shiflett, in developing this original method of orbital tracings, has made it possible for the ophthalmoplastic surgeon to approach the problem of orbital restoration with much more assurance and peace of mind than was formerly possible. The true extent and location of orbital injuries and deformities can now be accurately determined and charted prior to operation. This three dimensional tracing may be made a part of the patient's permanent record and is thus available in the operating room for reference at the time of operation. By knowing in advance the location and amount of correction required, the surgeon can proceed with the reconstruction with greater promise of success than has heretofore been possible, and by reference to such a diagram dangerous areas may be avoided. In some instances certain patients in which a cosmetic operation might result in fatality may be eliminated from surgical consideration entirely.

Transplantation of Human Vitreous. DR. NORMAN L. CUTLER, Wilmington, Del.

Thirteen cases of transplantation of vitreous were reported for hemorrhages into the vitreous in 8 and for detachment of the retina in 1. The results in 4 of the former were considered successful and in 1 partially successful. The outcome in 1 of the latter was considered successful. In all cases vision returned to the preoperative level. In the cases of hemorrhage no eye had less vision at the end of the procedure. In the cases of detachment 2 eyes had reduced vision, although 1 was expected to improve.

The improvement in vision in the cases of hemorrhage with a successful result ranged from light perception to 20/50—, and from 20/300 to 20/25. Although the operation was successful in but 1 of the 5 cases of retinal detachment, all these cases were considered difficult, and operations for retinal detachment had been performed previously.

DISCUSSION

DR. FREDERICK C. CORDES, San Francisco: As a civilian consultant in ophthalmology to the Surgeon General of the Army, it was my privilege to make observations in most of Dr. Cutler's cases, both before and after operation. In the first case it was of great importance to determine whether or not human vitreous could be transplanted successfully. From a surgical point of view the result was excellent; and but for a macular lesion, as Dr. Cutler has already pointed out, vision would have been materially improved.

Case 4 in the table was the third case in which an operation was performed, and it was probably the most spectacular. The patient was on the program for rehabilitation of the blind and, having finished his orientation training, was ready to be sent to Avon, Conn., to Old Farms, for his advanced training. The restoration of vision from 20/300 to 20/25 in six weeks was most dramatic, particularly after a period of eight months of almost complete blindness.

The result of vitreous transplantation in cases of retinal detachment has not been so good. However, in my opinion, not enough work has been done on this to justify condemnation of the procedure. The procedure may offer definite possibilities in carefully selected cases. It must be admitted, of course, that eight months, the longest interval after hemorrhage in the vitreous, does not exclude the possibility of further spontaneous clearing. Most ophthalmologists have seen cases in which as long as a year was necessary before complete absorption had occurred, and in many of these cases vision was again restored to normal. On the other hand, one has also seen cases in which at the end of that time organization of the hemorrhage had developed, together with retinitis proliferans. Experience has shown that in the latter cases little can be done once organization and retinitis proliferans have set in.

Of interest to me in these cases was the fact that for several weeks the clouded vitreous immediately surrounding the implanted vitreous contiued to clear rather rapidly, while the vitreous at the periphery, away from this area, showed no appreciable change.

Further experience will be necessary before it can be determined whether or not the risk involved in transplanting vitreous is small enough to make it advisable to use the procedure in case of hemorrhage in the vitreous, with the hope that it may prevent the organization that occurs in some of these cases. In addition, it will be important to determine whether or not some method of preserving vitreous can be devised, so that it can be available on short notice in cases in which its use may be indicated.

This paper must be considered a preliminary report, and further experience will be required before the use of, and indications for, vitreous transplantation can be evaluated. However, the work done by Dr. Cutler proves that human vitreous can be transplanted, without regard to blood groups.

Dr. Cutler is to be congratulated on this piece of interesting work, which required a good deal of courage to perform.

DR. JOHN MCGAVIC, M.D., Phoenixville, Pa.: It requires courage to report the first results of a new operative procedure, especially one which appears at first glance as bold as this. When a new operation is described, one can be certain that the original technic will undergo modification, that the indications will later be better defined and that there will be some resistance to the introduction of a new idea.

Ophthalmologists generally have such high regard for the vitreous that they avoid it whenever possible. We, therefore, owe Dr. Cutler our thanks for doing the pioneering work in the transplantation of human vitreous.

Dr. Cutler did some experimental investigation on animals along this line several years ago. Those who saw the eyes with which he has been working realize that they were damaged globes which had more pathologic changes than the hemorrhage into the vitreous and the detachment of the retina for which he did the tranplants. I have had the privilege of seeing several of the patients in Dr. Cutler's series and have dealt with many similar patients at another Army general hospital. I assure you that these eyes tax the ingenuity of any

ophthalmologist. Therefore, let us not be too critical of the visual result in these cases, but examine the technic of the procedure, its potentialities and the indications for using clear vitreous to supplant abnormal vitreous.

Dr. Cutler has pointed out that the blood groups of the donor are of no importance. The vitreous has no metabolism of its own and reflects chemical changes rather slowly. It is, therefore, rational to suppose that it can be transplanted.

It may be that aspiration of multiple small, nonmagnetic foreign bodies and aspiration of parasites from the vitreous will be carried out more often now that replacement of vitreous with vitreous has been proved feasible. I mention this because the incidence of foreign bodies and parasites has increased during the war, and they will be seen by many ophthalmologists in the postwar years.

In describing the operative procedure, Dr. Cutler mentioned withdrawal and injection of the vitreous in the equatorial region of the eyeball. One might well consider using the pars plana, which is avascular, or relatively so, to lessen the danger of hemorrhage and detachment of the retina. Although the lens will be close to the needle when the globe is collapsed, there should be little danger of injuring the lens, since the needle will be directed posteriorly. When the retina is already detached, it would seem advantageous to inject vitreous back toward it, rather than through a new hole in the retina made by the injecting needle. The pressing out of aqueous through the diathermy holes would be more effective, and perhaps more so if galvanic current, rather than the diathermy, were employed, as these holes stay open better.

Dr. Cutler has pointed out that attention to placing the clear vitreous in the visual axis is important. In the cases of hemorrhage in the vitreous, it would be most helpful if there were some means of foretelling the fate of the blood in a given eye. All have seen three types of reaction: complete absorption; partial absorption with retention of fluffy, cloudlike blood in the lower periphery of the vitreous over a protracted period, and organization of blood into bands. The first two types do not call for operation. Perhaps if one could foretell that bands would form—and this may be due to concurrent inflammatory reaction—the transplantation could be done while the blood was still loose. After the bands have formed, it is impossible to do more than clear the area between bands; and one may be afraid to do this lest the retina become detached by traction during temporary hypotony.

Spontaneous hemorrhage in the vitreous in young men does not seem to me to be an indication for this procedure. I saw a rather high percentage of patients with Eales' disease in the Army, and I cannot recall any who did not have spontaneous absorption, with normal vision and a normal fundus. I do not refer to patients who have sustained injury or to eyes complicated by perivasculitis, uveitis or diabetes with hemorrhage into the vitreous. The procedure described does not affect the underlying cause of the hemorrhage, which is not known, and would not, therefore, present the recurrence

of hemorrhage. It might result in less active absorption of blood when recurrence does take place.

Dr. Cutler reports discouraging results in cases of detachment of the retina. It should be reemphasized that these were cases of severe detachment, complicated by other factors, including previous diathermy. I believe there is reason to be more optimistic about using vitreous instead of air or saline solution. The maintenance of normal intraocular pressure is gratifying. In 1 of Dr. Cutler's 5 patients the result was spectacular. The role of the vitreous in the production of so-called idiopathic detachment is important but the mechanism has not yet been fully clarified. It will be interesting to note whether or not the foreign vitreous shrinks or remains detached from the native vitreous. One may learn as much about the mechanism of idiopathic detachments as one gains in therapy with this procedure.

To those who plan to employ this procedure, it is suggested that it be tried on animals first. Vitreous is tricky to handle, and only enough for one injection may be obtained from a donor globe. It would be embarrassing to be ready to inject donor vitreous and have something go wrong. Some of us at Valley Forge General Hospital used rabbits and utilized human eyes which had to be enucleated to develop our technic. We did not get beyond this, but we did find that we were glad to have practiced on animals and on eyes which had to be enucleated, rather than use the vitreous in clinical cases first.

We are indebted to Dr. Cutler for this fine presentation of another of his truly original pieces of clinical research. We hope that he will go ahead and will present the chemical, physical and histologic pictures resulting from transplantation of vitreous.

Dr. John M. McLean, New York: I should like to ask Dr. Cutler to enlarge further on the advantages of maintaining that 1 per cent of normal chemical constituents of the vitreous in the material which is put back into the eye, or is put into the eye, and its advantages as compared with those of saline or other physiologic solution.

I ask this because about three years ago, at New York Hospital, I had an unusual opportunity to transfer vitreous, and I should mention that this was done at the suggestion of Dr. G. J. Bertrand, who was then resident. The patient, a man with known atrophy of the optic nerve, had an injury to one eye, with a large hemorrhage in the vitreous, which would not absorb. His other eye was completely blind from the optic nerve atrophy and had no light perception. In that patient there was the rather unusual opportunity to tranfer vitreous from one eye to the other eye of the same patient. It was done in this fashion: The vitreous was removed from the blind eye first and replaced with a saline solution; then it was used to replace the bloody vitreous in the other eye, thereby eliminating the various potential difficulties of tranfer from one person to another. The result was not too satisfactory, and in the follow-up examination the visual result was not at all satisfactory. In the follow-up observation of the state of the vitreous over a long period, the eye in which vitreous had been replaced with saline solution seemed to have clearer and more satisfactory media than the eye in which vitreous had been replaced by the same patient's clear vitreous. The vitreous bodies in the two eyes

were equally fluid. That is what makes me wonder whether one has an adequate chemical reason in cases of detachment for switching from injection of saline solution to that of vitreous to restore the position of the retina. Certainly, when one passes normal human vitreous through a needle even once, much less twice, one has thoroughly destroyed most of its physical properties (Friedenwald, J. S., and Stiehler, R. D.: Structure of Vitreous, ARCH. OPHTH. **14:** 789-808 [Nov.] 1935).

DR. D. K. PISCHEL, San Francisco: I had the opportunity to examine 2 of the patients on whom Dr. Cutler had performed vitreous transplantation, both with retinal detachment. I examined the vitreous by means of a Koeppe flat contact glass and the slit lamp. In 1 of these patients the vitreous cavity showed no signs of any vitreous structure. In other words, it was an optically empty space, like the normal anterior chamber. I suppose that the vitreous which had been injected, as well as the original vitreous, had become entirely degenerated and liquefied, so that a uniform condition prevailed throughout the vitreous cavity.

In the second case, similar examination with the slit lamp and contact glass showed a small mass of fairly normal-looking vitreous immediately behind the lens. The posterior two thirds of the vitreous cavity, however, was optically empty, as in the first case, suggesting, again, liquefaction of the injected vitreous with a high degree of shrinkage of the original, or native, vitreous. Both eyes were quiet and showed no sign of irritation from the operation.

I should imagine, as Dr. McLean said, that any vitreous injected through a hypodermic needle would be considerably altered and, even if slightly gelatinous to start with, would be fluid after it had been injected.

The fact that the eyes stood the operation without any sign of inflammation is a favorable sign, and should lead one to think that this rather radical-sounding operation was not so radical after all and that ophthalmologists are justified in trying this new method whenever possible.

DR. NORMAN CUTLER, Wilmington, Del.: Perhaps I should state that I do not know the answers to some of the questions. The withdrawal of vitreous and the injection of saline solution have, of course, been performed by many surgeons. Zur Nedden reported on approximately 300 cases, although his data were not detailed enough to give one a clue to the possibilities.

It was not my purpose, in attempting this procedure, to stress the possibility of injecting vitreous rather than saline solution; rather, I wished to see whether vitreous could be injected and what the result would be. I am of the opinion that the 1 per cent protein in the vitreous is important. I do not know why, and I do not think that one is in a position to say. There are so many things about the vitreous which one does not know, except perhaps to be a little afraid of it; I feel that an attempt should be made to know more about it.

Dr. McLean suggested that the effect of passing vitreous through a needle was to destroy its structure. I am sure it is not known whether that is actually so. After all, the structure is ultramicroscopic. One needs to know what happens to cause vitreous to change from a sol-gel state to a sol state.

With regard to the observations of Dr. Pischel, which we were happy to have, the fact that the vitreous was injected and apparently became fluid was fairly evident, and it was confirmed in the few cases in which I was able to use a flat contact lens. What the condition of the vitreous was before other vitreous was injected I do not know. It was certainly abnormal, but how fluid it was could not be determined. Whether the viscosity of the vitreous can be maintained after it is subjected to some trauma, which is present in a transplantation procedure, remains to be determined. I was, of course, careful to point out that this was not my hope in this procedure; in fact, some of the vitreous which I injected was fluid at the time I injected it.

FOREIGN BODIES OF THE GLOBE: A SYMPOSIUM

Pathologic Aspects. MRS. HELENOR CAMPBELL WILDER, Washington, D. C.

Retained foreign bodies were found in 731 of 3,882 eyes of soldiers studied at the Army Institute of Pathology during World War II. All globes with penetrating wounds were examined roentgenologically and were searched for foreign bodies. In many instances the particles were so small or so deeply embedded in organizing hemorrhage or inflammatory membrane that they were not recovered from the gross specimens and became visible only on microscopic examination. These could not be subjected to the magnet test, but sections containing them were stained with prussian blue. The particles which gave a positive reaction for iron were classified as probably magnetic, although the degree of their magnetic quality could not be determined, whereas those giving no reaction were regarded as nonmagnetic. In many eyes the particles were multiple, and in a few both magnetic and nonmagnetic materials were observed.

The nonmagnetic foreign bodies comprised a variety of materials. Among the metal substances, copper, brass, lead, nickel and zinc alloy were identified; among the nonmetallic, glass, vegetable matter, eyelashes, rock, shale and clay predominated.

Abscesses were frequently seen around the sites of foreign bodies of various types, being almost invariably present around vegetable matter. In the vitreous chamber the subsequent formation of a pyogenic membrane resulted in retinal detachment and phthisis bulbi. Foreign body granulomas appeared around all kinds of foreign bodies, but the formation of giant cells seemed to be stimulated particularly by eyelashes and vegetable matter.

Sympathetic uveitis developed in 5 eyes with retained intraocular foreign bodies. A variety of infections followed introduction of foreign material into the eye. Masses of cocci, bacilli and unidentified fungi were sometimes seen in abscesses in the vitreous.

The paper was illustrated with thirty-five lantern slides.

Experimental Work on Intraocular Foreign Bodies. LIEUTENANT COLONEL G. C. STRUBLE, Medical Corps, Army of the United States.

The author's method of removing magnetic foreign bodies from the eye with the Lancaster magnet was published in the *American Journal of Ophthalmology* (**29:** 151-161 [Feb.] 1946). Further clinical experience and experimental studies, using the Lancaster hand magnet with a foot switch, were made to determine the reaction of metallic foreign bodies to the magnet current within the eye. Experiments were carried out on eyes of freshly killed hogs less than twelve hours dead which were kept refrigerated. Observing the metal particles in the vitreous under magnetic attraction showed that success of the operation on the first application of the magnet depended on the initial impact of the foreign body against the inner layers of the globe. Larger fragments were delivered without difficulty. Small fragments 0.25 mm. and less in size had to be aided in their path through the uvea.

Foreign bodies were experimentally planted in the retina and the choroid, and efforts to remove them with a magnet were made in the usual manner. Where the fragment was pulled laterally for a distance of 3 or 4 mm. toward the sclerotomy opening and magnet, a tear or gutter in the retina or choroid was created throughout the length of the movement of the particle. When the foreign body was embedded in the choroid or the retina directly overlying the scleral opening, it was always pulled through by the magnet on the first attempt, with a minimum of trauma. This observation is further argument for precise, pinpoint localization of a metallic foreign body embedded in these structures at the time of surgical removal.

Small particles free in the posterior portion of the vitreous can be brought forward temporarily but retract into the vitreous as the current is turned off. None could be drawn into the anterior chamber, and none would become embedded in the ciliary body. Particles 1 mm. in size and larger could be consistently brought forward. They would move slowly but could be embedded in the ciliary body or drawn into the anterior chamber.

Concerning magnetic foreign bodies in the retina, particles of less than 1 mm. in size were not disengaged from the retina, but those 1 mm. and over were suddenly disengaged and moved rapidly through the vitreous toward the magnet. The larger the particle, the greater the impact on intervening tissues. All particles except those 0.25 mm. in size caused some separation of the retina.

Attempts to remove particles planted between the retina and the choroid were unsuccessful if they were less than 1 mm. in size. Particles 1.5 mm. or larger could always be delivered. The larger the foreign body, the more rapidly it would perforate the retina. Disengagement was always sudden and under great tension.

Foreign bodies placed between the choroid and the sclera were most difficult to remove. Particles 1 mm. and less in size could not be removed, but particles 1.5 mm. and over produced an increasing degree of choroidal separation and retinal elevation. In no instance were the choroid and retina perforated or the foreign body delivered.

It was realized that many factors existed in these experiments that do not exist in the human eye. The author stated, however, that given

the problem of removing a foreign body, with identical equipment available, it is logical to assume that the results obtained experimentally represent the best which can be expected.

Roentgenographic Localization of Intraocular Foreign Bodies. DR. W. H. DROEGEMUELLER, Chicago.

To us, as ophthalmologists, the problems in the localization of intraocular foreign bodies involve more than the purely technical aspects of the methods of localization now available. The roentgenologic technics of localization and the calculations used are highly perfected, with little call for improvement unless it would be in the better visualization of the less opaque foreign bodies. However, the ophthalmologist, in transposing the position of a foreign body from the roentgenologist's chart, is subject to more or less error. Whether the Sweet method, the Comberg contact lens technic or markers of various types sutured to the exposed portion of the eye are used, the basis for the localization is the relation revealed between the foreign body and the orientation-marking device, which has a known position to the eyeball proper.

The checking of the primary localization can be accomplished by a method described by Colonel Struble in 1942 as pinpoint localization. Absolute localization is essential in any attempt to remove a nonmagnetic foreign body, and usually the first attempt is justified only after a careful consideration of the findings in the localization procedure. In the usual cases of magnetic foreign bodies, in which magnetic power can make up for inaccuracies of localization, the recovery of the foreign body must be considered in the light of the trauma produced in the extraction. An argument in favor of accurate localization is the hypothesis that the least traumatizing pathway of removal would be directly out of the eyeball, without the production of any lateral drag on the retina or the choroid. But this is a hypothesis, and there are no cases in which the foreign body could be drawn away from the retina with less harm than through it. (By means of lantern slides the author illustrated several methods of localization of foreign bodies by use of specially applied markers attached to the eyeball. The Berman localizer reveals the pressure of magnetic and some of the larger nonmagnetic fragments by an intensity sound signal. It is not as sensitive as the roentgenographic method.)

Parallax studies and the technic of injecting air into Tenon's capsule, as described by Spackman, deserve special mention. The latter procedure is of use in determining whether a foreign body is intraocular or extraocular. The routine use of stereoscopic plates has a definite value. The ophthalmologic problem in roentgenologic localization of foreign bodies is in critically evaluating the technic used and in considering the probable trauma to the eye in the process of extraction.

Surgical Removal of Intraocular Foreign Bodies DR. TRYGVE GUNDERSEN, Boston.

A silhouette of the human body covers a surface of about 4.2 square feet (3.9 square meters). The extremities occupy about 35 per cent of the area of the silhouette; the head and neck, about 13 per cent,

and the eyes, about 0.1 per cent. Nevertheless, the percentage of battle injuries of the eyes is ten to fifteen times as high, owing to the fact that a small abrasion of the cornea will cause a soldier to be evacuated. In the eighty-four hospitals in the African and European theaters the cases of ocular injuries constituted about 2 per cent of the hospitalized patients. In the entire war, about 1,400 Army and Navy service men were made blind by battle injuries.

Some new features of ocular injuries were learned in the war. The velocity of fine fragments was found to be much higher than in the first world war. A single wound of entry with multiple intraocular foreign bodies was thought to be due to molten metal. New instruments for location of foreign bodies (Berman; Carney) were introduced. Magnets of various types (Lancaster, Mellinger and others) were used extensively.

Factors which influence the choice of operation for intraocular foreign bodies are the amount of damage to the eye, the nature of the foreign body and the possibility of removal of the foreign body. Because of the difficulty in determining damage to the eye borderline injuries should be observed for a time before enucleation is decided on. Severe hemorrhage into the anterior chamber usually becomes associated with glaucoma and calls for early removal of the eyeball. Foreign bodies differ tremendously in their toxicity. Gold, silver and platinum are tolerated fairly well; lead and zinc, less so. Soft iron and copper, and particularly brass, are tolerated poorly. "Plexiglass" seems to be relatively inert.

Practically no intraocular magnetic foreign bodies defied removal with the modern methods of localization and approach. Nonmagnetic foreign bodies require meticulous localization. They can be removed with ingenious devices, such as Thorpe's endoscope or under direct observation. Both the anterior and the posterior route were used for removal of magnetic bodies. The anterior route, when feasible, was considered safer.

Reconstruction of the Eyelid. MAJOR ARTHUR E. SHERMAN, Medical Corps, Army of the United States.

From the summer of 1944 to the spring of 1946, nine of the Army general hospitals in this country were designated as centers for plastic and ophthalmic surgery. Of the large volume of casualties evacuated to these centers for reconstructive surgery during that period, each center handled from 300 to 400 requiring ophthalmic reconstruction. In some of these hospitals this type of surgery was the function of the ophthalmologic service; in others, of the service of general plastic surgery.

Certainly, all of us who did much of this work are indebted to the late Dr. John M. Wheeler for the methods and principles which he developed and advocated. For vertical full thickness scars of the eyelid with notch deformities at the margin and for small full thickness loss of an eyelid, Wheeler's halving repair was the procedure of choice. For loss of skin from burns or lacerations, with resulting cicatricial ectropion or lagophthalmos, no better method of correction was found than that used by Wheeler, with the use of intermarginal lid adhesions, proper preparation of the bed for skin

grafting and the use of full thickness skin from the upper eyelid or posterior auricular skin for the graft.

All degrees of full thickness loss of the upper or lower eyelid were encountered. For loss of a large part of the tarsal portion of an eyelid, Dr. Wendell Hughes's method of reconstruction was very useful. In repair of some of the more extensive losses, it was necessary to use sliding skin flaps as a preliminary procedure, before joining the eyelids according to the Hughes method. Moderate or extensive loss of bone of the orbital margin was replaced by cancellous bone graft from the ilium. In some of these cases it was also necessary to enlarge the orbital socket with mucous membrane grafts. A number of illustrative cases were discussed, together with the methods used for reconstruction.

DISCUSSION

Dr. Edmund B. Spaeth, Philadelphia: Dr. Sherman has had a tremendous clinical experience in ophthalmic plastic surgery. I have seen some of his results, and it behooves us to profit from them. Differences of opinion are natural—perhaps even healthy. My discussion, while it expresses a difference of opinion on some points, in the final analysis simply supplements and emphasizes certain of his statements.

As to his dislike of the use of pedicle flaps, I see no way out of their use in certain cases. Underlying disease of the osseous structures and the presence of deep cicatrices make it almost impossible to utilize, with full satisfaction, free skin grafts, of either the razor-cut or the full thickness type. Dr. Sherman calls attention to his satisfaction with the use of supraclavicular skin—almost any amount of this can be easily and safely transplanted by means of a delayed transfer pedicle flap from that area to the region of the eyelids. Even greater, however, is his dislike of pedicle flaps from the immediate neighborhood of the eyelids. Given a patient with an intact eyeball and with an extensive deficiency of the conjunctiva, there is no better way of making the two transplantations simultaneously than that of lining a pedicle flap first with the mucous membrane and then transplanting both to the new position.

Slide 2 answers the problem of repair of loss of bone as the result of osteomyelitis, now quiescent, and sequestrectomy. Also, it shows my preference for the use of cartilage, in this instance an autogenous graft, though cadaver cartilage might have been as satisfactory, in contrast to Dr. Sherman's preference for cancellous bone from the ilium. Frankly, I have discontinued the use of autogenous bone grafts wholly, and for at least five years. This may be an error on my part, but it seems that the cartilage grafts have done as well.

I agree wholly and without equivocation with Dr. Sherman's statement in regard to pressure dressings and his disagreement with Brown in this respect. The sutures that Brown has used are not only unnecessary but unwise. They must be introduced; they must be removed; they are a wick for low grade secondary infection of the graft; they cause additional scarring, and they limit the size of the graft possible. They give a pressure dressing, to be sure, but one less satisfactory than that obtained with a mold of dental stent or

dental wax fitted to the site for the graft or grafts immediately prior to their placement in position.

Again, Dr. Sherman emphasizes the necessity of intermarginal adhesions with free skin grafts to the eyelids. I agree to this without argument when both the upper and the lower lid are corrected simultaneously. When, however, the upper lid only needs correction and the conjuntiva is adequate, intermarginal adhesions are not necessary.

Slide 3 shows such a case. It illustrates the ectropion residual to severe herpes ophthalmicus necroticans. The patient was blind in her opposite eye, and intermarginal adhesions would have limited her vision seriously during the process of recovery. The same objection applies to an isolated correction for ectropion in the lower lid if similar circumstances are present. Temporary intermarginal sutures must be employed during use of the postoperative dressing to protect the cornea from damage, but as soon as the dressings are permanently removed the sutures can also be removed.

Dr. Sherman gives credit to Dr. Wendell Hughes for a principle in total reconstruction of the eyelid which I also wish to mention. A tremendous amount of credit properly belongs to Dr. Hughes for the reconstruction he has done in these difficult cases. It in no way detracts from this credit to call attention to the fact that the underlying principles of this procedure were first presented by Landolt and subsequently extended considerably by Axenfeld. This criticism may seem trite, but it is proper. It was a pleasure to discuss this paper, and I compliment Dr. Sherman on his beautiful work.

Dr. James N. Greear, Washington, D. C.: The Army Medical Corps has, indeed, been fortunate in having available men with such thorough understanding of ophthalmic plastic surgery as Major Sherman's work so ably demonstrates. All are agreed as to our indebtedness to the late Dr. John M. Wheeler for the principles of this branch of surgery. Dr. Wendell Hughes has made a valuable contribution to reconstructive surgery of the lids, and the procedures advocated by him have proved extremely useful, especially when there has been extensive damage to one of the eyelids.

It has been my experience that there is more redundant skin on the eyelids of old people, thus making the procedures advocated by Hughes much more feasible than in the case of younger persons.

It is essential that uniform pressure be maintained over the grafted area in any plastic procedure. This is particularly true when free grafts are used. It is especially difficult to maintain such pressure after grafting an eyelid if enucleation has previously been performed. The method of applying pressure, as advocated by Dr. Barrett Brown, yields excellent results in such cases. Dr. Brown's procedure is to leave long the sutures around the margin of the free graft, tying these over mechanic's waste so as to produce uniform pressure.

There is certainly no more adequate operative measure for correction of notching of the lid margin than Wheeler's halving operation.

I should like to emphasize the importance of early treatment and care of severe injuries to the eyelids. Owing to the greatly increased activity of the air forces and the motorized units of troops in the recent war, there were large numbers of patients with severe burns of the

face. These patients were given emergency treatment in evacuation or station hospitals and soon were returned to general hospitals, where they received definitive care. It is generally recognized that the most serious sequela of such an injury is damage to the eye from exposure. This was inevitable unless preventive measures were instituted at an early stage. In order to forestall cicatricial ectropion, with resultant exposure keratitis, one of two procedures was generally carried out in the general hospitals in the European Theater of Operation.

As soon as severely burned lids began to granulate, a split thickness skin graft was placed over the raw surface; this served as a dressing graft and prevented extensive contracture of the lids. A dressing graft may be taken from another person, in which case it serves for a limited period only. Such a graft was necessary in exceptional cases in which it was felt that further loss of skin would be detrimental to the patient. This procedure was helpful, and no doubt it was responsible for preventing serious damage to the eyes of many patients. Even if the patient was most severely burned about the face, there was seldom serious damage to the lid margins; therefore, intermarginal adhesions were feasible in these cases. The formation of these adhesions at an early stage prevented the severe cicatricial ectropion which was certain to develop. This procedure could be followed by early grafting if extensive damage to the skin and subcutaneous tissue predicted extensive cicatricial contracture as an end result. These temporary grafts could be replaced by more suitable skin later if need be. This method proved to be eminently satisfactory and provided adequate protection to the eye.

In most instances cicatrization could take place and a suitable skin graft to the lids be made at a later period, the eye having been well protected in the meantime.

Many of the patients receiving such severe burns of the head had no available skin on the eyelids or the postauricular area for covering the eyelids as a final operative measure. In such cases free grafts from the supraclavicular area proved to be satisfactory. It was found that early repair of damage to the eyelids would in many instances save the patient weeks of hospitalization. Even when there was loss of more than one third of the eyelid, the defect could be corrected by utilizing the sliding flap from the temporal side. This could be carried out as a primary measure or after débridement and secondary closure, even though a period of from three to five days had elapsed since the original injury to the lid. This procedure was particularly applicable to injury to the lower lid.

If such complete repair of injury to the eyelid was not feasible, a marginal suture was placed wherever possible in order to preserve the lid margin. This was a distinct aid in subsequent reconstruction of the lid.

In reviewing the recent literature dealing with surgery of the eyelid, one is impressed with such expressions as "marked tendency to tissue separation and frequent recurrences of the initial defect." Such an occurrence should be rare if one of the most important principles of plastic surgery is observed, that is, that the suture lines should never be closed under tension.

DR. ARTHUR E. SHERMAN, Newark, N. J.: There would, of course, be a difference of opinion as to some of these procedures. I did say in my paper that one should avoid pedicle flaps if at all possible. I did not mean that there are not at times cases in which one must use them.

With regard to bone grafts and cartilage grafts, again, there is great difference of opinion. My associates and I used cartilage grafts, both autogenous and preserved cartilage; and we were not as well satisfied with the cartilage as with cancellous bone from the ilium. I feel that the advantage of cancellous bone is that it is easily shaped to fit the defect. Within two or three weeks it is firmly attached; that is, it has grown fast to the adjacent bone, and within another month or two the bone graft has been covered with a pseudocortex. The absorption of the graft is negligible.

I have never felt that dental stents should be used with a full thickness graft on the eyelid, but there is difference of opinion with regard to that.

I still believe that lid adhesions are extremely important if one wishes to obtain the most uniformly good or excellent result. The adhesions serve an important purpose in that during the period from about the second to the eighth week after operation these full thickness grafts, or Thiersch grafts, will tend to contract, but, with the constant pull of the opposite lid, through the adhesions, the grafts will become smooth and fine textured, so that at the end of three months, when the adhesions are cut, the graft is usually the size it was originally and there should as a rule be no residual slight ectropion. I realize that Dr. Hughes's operation is a modification of older procedures, but it is a definite modification. I believe that it is more useful than some of the older procedures, although there are times when some of the older procedures have their place.

With regard to Dr. Brown's method of using pressure on the eyelid, that, again, I think is simply a modification of the Gillies form of dressing, in which he used an epithelial "outlay." In the hands of such surgeons as Dr. Brown, the method usually yields fairly good results, though I believe less uniformly; certainly, the average ophthalmic plastic surgeon does obtain much more uniformly good results by following Wheeler's perfected procedure.

I am glad that Dr. Greear brought out the use of lid adhesions and that of the Thiersch graft in the immediate care of these burns overseas. I can recall several men who were evacuated to us on whom nothing more than one or two lid adhesions on each side had been produced, and who three months from the time they were burned, required no additional skin grafting. All that one had to do was to cut the lid adhesions and the lids were perfectly normal.

Roentgen Therapy of Retinal Diseases Characterized by New-Formed Blood Vessels (Eales's Disease, Retinitis Proliferans): A PRELIMINARY REPORT. DR. JACK S. GUYTON, Baltimore, and DR. ALGERNON B. REESE, New York.

This article will be published, with discussion, in a later issue of the ARCHIVES.

Marginal Corneal Infiltrates and Ulcers. Dr. Phillips Thygeson, San Jose, Calif.

Simple catarrhal ulcers and infiltrates were the commonest type of corneal disease observed in this study of 200 cases of marginal ulcer. Although usually benign, the ulcers were sometimes severe and recurrent, leading to cicatrization extending into the pupillary area or to irregular astigmatism. They were almost always associated with chronic catarrhal conjunctivitis, usually staphylococcic, but in a few cases caused by other such agents as the diplobacillus of Morax and Axenfeld, the nonhemolytic streptococcus and coliform bacilli. In 2 cases ulcer was associated with vernal conjunctivitis, and in 1 case it was an allergic reaction to a local anesthetic. Coincidental blepharitis was an almost constant feature of the cases of the staphylococcic and diplobacillary type. Marginal catarrhal ulcers were also associated with acute catarrhal conjunctivitis caused by Staphylococcus aureus and by the Koch-Weeks bacillus (Hemophilus conjunctivitidis). In 4 cases the ulcer occurred coincidentally with gonorrheal arthritis and was presumably secondary to metastatic gonorrheal conjunctivitis. In 8 cases it was secondary to chronic conjunctivitis for which the cause could not be found.

There were 14 cases of ring ulcers and infiltrates, which, with 2 exceptions, appeared to be endogenous, occurring in association with gonorrheal arthritis, bacillary dysentery, influenza, periarteritis nodosa and lupus erythematosus. The 2 exceptions were cases of severe staphylococcic conjunctivitis with multiple marginal ulcers which coalesced to form ring ulcers.

There were 6 cases of chronic serpiginous (Mooren's) ulcer, typical in clinical appearance and course. There was no associated conjunctivitis in these cases and, so far as could be determined, no relation to any systemic infection. Careful laboratory studies of smears, scrapings and cultures failed to reveal any significant micro-organisms, and animal inoculations all gave negative results.

Prophylaxis of recurrent marginal ulcer of the catarrhal type is believed to be entirely a matter of controlling the chronic conjunctivitis. This, in turn, is largely dependent on control of the chronic blepharitis, either staphylococcic or diplobacillary, which in most instances was found to be the primary focus for both the conjunctivitis and the keratitis. No information of prophylactic value was obtained in connection with either the ring ulcer or the chronic serpiginous (Mooren) ulcer.

Treatment of catarrhal marginal infiltrates and ulcers appeared to consist entirely in treating the primary conjunctivitis and blepharitis. Except in rare instances, local treatment of the ulcer was unnecessary. Staphylococcus toxoid proved to be of value in preventing recurrence of catarrhal ulcers, even when cure of the primary conjunctivitis was not obtained. Treatment of ring ulcers and infiltrates was also directed toward the primary cause, which was almost always a systemic disease. Local treatment was found to have little value, and cauterization of the ulcer seemed only to aggravate it. In the treatment of chronic serpiginous (Mooren's) ulcer the sulfonamide compounds and penicil-

lin were ineffective. Delimiting keratotomy and repeated paracenteses were apparently successful in arresting progression in 3 of the 6 cases.

No evidence was obtained to indicate that riboflavin deficiency contributed to the development of marginal ulcer or that treatment with riboflavin was curative.

No true example of marginal keratitis due to acne rosacea was seen in this series. There were 9 cases of rosacea with typical catarrhal marginal ulcer, but in these cases the ulcer was believed to be due to secondary staphylococcic infection.

DISCUSSION

DR. C. L. SCHEPENS, Brussels, Belgium: I congratulate Dr. Thygeson on his excellent paper. Personally, I learned a great deal from it.

The geographic distribution of corneal infections due to the Koch-Weeks bacillus is of interest. It was frequent in Western Europe thirty years ago. Nowadays, it has practically disappeared. I believe that at Moorfields Hospital (London) there has not been a case in fifteen years. Personally, I have never seen a case in Brussels. The same comment applies to vernal conjunctivitis, which is frequent in Spain but is seldom met with in Belgium, Netherlands or France.

It has been stated that penicillin is not active against the diplobacillus. I believe that some authors have found it active (F. A. Juler, London, and others). Perhaps further investigation will have to be made on this particular point.

I have never had any striking results with vitamin A, thiamine, riboflavin or ascorbic acid. I have used vitamin A locally and in intravenous injections. One has to be cautious with the injections, which sometimes cause diarrhea. I have tried intravenous injections of riboflavin, following the advice of Williamson-Noble (London), but with no favorable results. Intravenous injections of 500 mg. of ascorbic acid, every other day for about two weeks, appeared more satisfactory.

One point of importance is the advisability of using atropine in every case of corneal involvement. It has been said that it is not necessary to use atropine in certain cases of corneal ulcer. This is contradictory to the teaching in many European universities. I believe the classic teaching is that atropine should be used when there is corneal involvement. If no atropine is necessary, it means a substantial shortening of the period of incapacitation. The advisability of using atropine depends, in many cases, on whether or not one believes that it has a favorable action on the nutrition of the cornea.

I agree with Dr. Thygeson that cauterization is sometimes done much too freely. In cases of stubborn corneal ulcer, it often does more harm than good.

In a difficult case of marginal ulcer, the following therapeutic means are worth trying, although they are not always successful. First, some marginal ulcerations heal more quickly with physostigmine than with atropine. Second, local applications of ultraviolet rays may be useful. The effect is enhanced if 1 per cent fluorescein sodium is instilled immediately before the light treatment. Third, the local application of beta rays by means of 10 mg. of unscreened radium sometimes gives good results. Fourth, small doses of roentgen radiation may also be used. One applies locally between 1/3 and 1/50 and erythema dose every

other day for six to ten days. This method sometimes gives good results in cases of ulcer following acne rosacea and in cases of Mooren's ulcer.

The general health of the patient must be cared for and much attention should be paid to his diet and to whether he takes enough fresh air. If the patient does not do well I like to take him to the hospital at an early stage, for general rest is important. I give him milk injections, and if he does not improve I do a tarsorrhaphy. I believe this operation is of value in every difficult case of corneal ulcer. I prefer it to a conjunctival flap, for one does not see what happens under the flap and in some cases the flap remains permanently adherent to the ulcerated cornea and has to be removed surgically after the ulcer is healed.

I am not in favor of paracentesis unless the tension is high. As a general rule, when there is a bad ulcer, I avoid surgical intervention on the cornea proper.

Dr. James H. Allen, Iowa City: My experience with marginal lesions of the cornea has been similar to that expressed by Dr. Thygeson. However, there are some points on which he and I differ, and I should like to speak about a few of these, as well as some on which we agree.

I think that staphylococcic marginal ulcers of the cornea should not be taken lightly, for several reasons. One is that I saw an eye destroyed by a lesion which began as a simple staphylococcic marginal ulcer and spread, formed a ring ulcer and produced central necrosis of the cornea, with rupture of the cornea and loss of the globe. That occurred in the days before antitoxin was used, and before sulfathiazole and penicillin were available.

However, another reason for not taking these lesions lightly is that at times they undergo exacerbations even under treatment, and one must not administer toxoid without caution. If one is dealing with a staphylococcic marginal ulcer of the cornea, it is better to give antitoxin until the ulcer is healed before beginning active immunization with toxoid.

I should like to show slides from 2 cases of staphylococcic marginal ulcer of the cornea. Infiltrations are seen at the upper nasal margin. The surface is ulcerated over these infiltrations. The patient was given 10,000 units of staphylococcus antitoxin intramuscularly daily for four days, with the result that the ulcerations healed rapidly. She was later immunized with staphylococcus toxoid, and no recurrences developed.

There is importance in emphasizing antitoxin treatment, even though penicillin is available. Dr. Thygeson has reported from his experience that 20 per cent of ocular strains of staphylococci do not respond to penicillin therapy. I can support that statement. However, it is my impression that more than 20 per cent of ocular strains of staphylococci are not affected by penicillin; therefore, antitoxin therapy should be considered if penicillin is not giving results in the treatment of staphylococcic marginal ulceration of the cornea.

I have seen numerous cases of superficial punctate epithelial keratitis as a result of vernal conjunctivitis, particularly the limbic variety; but I have not seen marginal corneal ulcer or central corneal ulcer with vernal conjunctivitis except in cases of secondary infection. In

a case of a youngster, severe marginal keratitis with ulceration was superimposed on limbic vernal conjunctivitis after he had been vaccinated.

My experience with acne rosacea has been similar to that of Dr. Thygeson. The ulcerated lesions have always yielded pathogenic organisms.

DR. PHILLIPS THYGESON, San Jose, Calif.: I wish to thank the discussers for their valuable comments, and I should like to discuss one or two of them.

Dr. Schepens mentioned atropinization. I have felt that in cases of simple marginal ulcer with little irritation of the iris the incapacity caused by the atropine outweighed the advantages. I have frequently substituted homatropine for atropine in such cases.

Dr. Schepens' experience with the Koch-Weeks organism in Europe has paralleled mine in this country. In my experience, the Koch-Weeks bacillus in conjunctivitis of epidemic form has been encountered only in Florida and California. I actually saw no case of this infection in New York in a period of about six years, although Dr. Weeks made his original studies on the organism there.

I shall certainly try roentgen rays and radium in treating Moooren's ulcer, in view of my only partial success with other methods.

Time prevented me from discussing the shock therapy of ulcer. It proved of definite value in my series of ring ulcers.

Dr. Allen's work on staphylococcus antitoxin in treatment of staphylococcic ulcers is outstanding. It is true that penicillin-fast organisms will become more frequent; and, whereas new antibiotics may be developed, it is certain that there will always be cases in which a staphylococcus antitoxin will be of great value.

Nonperforating Ocular Injuries in Soldiers. DR. BENJAMIN RONES and MRS. HELENOR CAMPBELL WILDER (by invitation), Washington, D. C.

This report dealt with 104 cases of nonperforating ocular injuries in soldiers. Forty-seven eyes were injured during combat or training. The interval between injury and enucleation varied from less than one day to two and one-half years. In these eyes hemorrhage was the condition most frequently encountered although nonpurulent endophthalmitis, detachment of the retina and secondary glaucoma also were often noted. Injury to the eyes of 57 soldiers had preceded induction into the Army. The shortest interval between injury and enucleation in this group was four years and the longest twenty-eight years. Chronic endophthalmitis was the most frequent lesion, with cataract, detachment of the retina and choroid, and secondary glaucoma following, in that order. A great variety of degenerative and late inflammatory changes were recorded.

An inflammatory reaction was present in 20 cases in which no operation had been performed. Purulent endophthalmitis in 2 of these cases had resulted from intraocular extension of purulent keratitis. A granulomatous inflammatory reaction was observed in the ciliary body and processes in a third case. The most striking cellular response was observed in a fourth case, in which there was active endophthalmitis, with dense lymphocytic infiltration around Schlemm's

canal and chronic inflammatory cell infiltration in the iris, ciliary body, retina and nerve head. This reaction was regarded as a response to infection. In the remaining 16 cases the inflammation was clearly a response to tissue trauma and subsequent hemorrhage, although in 2 cases the number of eosinophils in the infiltrate suggeseed an allergic factor. The inflammatory process was chiefly in the anterior segment in 12 cases and in the posterior segment in 2 cases. The paucity of the cellular response was noteworthy; lymphocytes and plasma cells were the dominant types. On the other hand, the exudative response was pronounced and resulted in the formation of anterior and posterior synechiae, together with pupillary and cyclitic membranes.

DISCUSSION

DR. W. I. B. RIDDELL, Glasgow, Scotland: In opening the discussion, it is difficult not to become lost in details of particular cases. When I heard that I was to take part in this discussion, I had only the title of the paper, and I wondered what I could contribute that might be of interest and bring a slightly different point of view to the consideration of the many problems which arise from this material.

It seems to me that the general military experience in the British army is similar to that in the American army, with no racial discrimination with respect to booby traps or grenades, but there is a difference in the injuries occurring in British civilian air raids because there destruction was indiscriminate; that is, the age groups represented the whole populaton, and the sexes were affected equally. One should not keep this in mind when considering the material at Washington, since these ocular injuries respond differently in older age groups than in younger ones and there might well be a sex difference also. One is aware of the remarkable sex difference in Eales's disease, which occurs predominantly in young men.

Another difference in the civilian ocular injuries was the enormous excess of those due to glass. In the large majority of cases in the heavy raids, the eyes were badly damaged or destroyed by flying particles of glass. The tissue reactions which my colleagues and I saw were mainly intraocular hemorrhages. We did learn to what a remarkable extent an eye can wtihstand a spicule of glass, the glass coming out and the eye being repaired. The injury does require surgical treatment.

One of the difficulties was that these civilian injuries were not emergencies. They were dealt with by general surgeons, and the patient might lie for twenty-four or forty-eight hours before any one discovered that there was anything wrong with the eye or that anything, in fact, could be done for him.

I recalled that before the war one of my colleagues, Dr. A. M. Wright Thomson, had wondered about the effect in eyes damaged in crush injuries. He is a careful observer, and negative findings are not always made by a careful observer. He collected 11 cases of crush injuries. These were mostly cases of young people who were crushed in elevators or between roller bands; that is, their chests and bodies were crushed. They all had subconjunctival hemorrhages. Many of them had subcutaneous hemorrhages around the eyelid, but of the 11 cases there was only 1 in which a strip hemorrhage was observed

in the retina. That negative finding occurred to me to be of importance, because it shows that an injury around the globe or its adnexa is required to affect the globe itself. About half the patients recovered, and in none of them did any injury to the bursa, psychiatric disturbance or arthritis develop. That point struck me as I was listening to the presentation of the paper by Dr. Rones.

When I looked at the material before the paper itself was presented, an amusing idea struck me, and I considered it more carefully. I thought it might be valuable in presentation not only of this material but frequently of clinical material if one divided the cases into arbitrary categories of one week, three months, a year, three and one-half years, and so on, presenting it in tabular form and concealing certain information, which might be brought out by a mathematical device. If there is one thing the armies and the government service in both Britain and America inquire into carefully, it is the report. They are always looking for dates and such things. I am quite sure that Dr. Rones knows the day of injury and the precise day of removal of the eyes. If one counts up the total number of days, calls the day of injury zero, or V-Day, ten days later, V plus 10, and so on, one may then use the logarithm of the days and get categories in which one can place the material, thereby spreading out the tabular material. I don't know whether I have managed to put that idea across to you. Perhaps you may think it is a silly sort of idea, but the table which Dr. Rones has in the text of his paper suggested it to me and it is a convenient and useful device for any series of clinical observations covering a long period. Arranged in any other way, the material makes an enormous table, which nobody can understand and few people read.

Another matter which came to my mind was the point brought up by my senior assistants, Dr. I. C. Michaelson and Dr. George I. Scott. In those days they were Brigadier Scott and Major Michaelson. They wrote a paper which appeared in the *British Journal of Ophthalmology* (**30**:42 [Jan.] 1946), dealing with 301 battle casualties, in 47 of which the injury was nonperforating, similar to those of Dr. Rones and Mrs. Wilder. They made a point which I think is of interest in considering material of this kind. They suggested that there is a difference between an air-conducted and a tissue-conducted injury. In general, an air-conducted injury affects the anterior segment of the eyes, and a tissue-conducted injury, the posterior section of the eye. Of course, there are exceptions; but in general this relation emerges from their material. In listening to the paper this afternoon, I got the impression that the same factor was present in this material.

DR. S. RODMAN IRVINE, Beverly Hills, Calif.: The value of the contributions Dr. Rones and Mrs. Wilder have made to knowledge of ocular pathology resulting from contusion injuries lies particularly in their analysis of the condition of the eyes with respect to the interval of time following injury. An experimental study on animals was made by Dr. George Kilgore, who produced varying degrees of contusion injury in monkeys and made an extensive study of the pathologic changes at various times after injury (Kilgore, G.: *Am. J. Ophth.* **25:** 1095-1099 [Sept.] 1942). The present authors have made a some-

what similar study on the human eye, taking advantage of the large series of eyes removed after injury, available at the Army Institute of Pathology.

One is impressed by the extensive damage that may occur to the eye as a result of a concussion or contusion injury; this, I believe, is not generally appreciated. Clinically, there is no doubt that one is often deceived by the appearance of an eye immediately after such an injury. It is only when recurrent hemorrhages take place and secondary glaucoma develops that the clinician is aware that a major disaster has occurred.

I wonder whether the authors encountered anything in particular that would lead them to predict that an eye would have recurrent hemorrhages, how soon blood staining of the cornea might occur if blood were allowed to remain in the anterior chamber in a glaucomatous eye and how soon cupping of the disk might be expected. Dr. Rones mentioned that in 1 case cupping occurred about six weeks after injury, but I wonder whether there are other cases in which the time interval might have been more definite. These are important questions to the clinician who desires to know how much leeway he has before it is necessary to evacuate the anterior chamber and remove the blood.

It is interesting to me that there was no incidence of sympathetic ophthalmia in spite of there being tremendous disorganization of the ciliary body and pigment throughout the globe. This confirms the observation that a perforating injury is necessary for sympathetic ophthalmia to develop, and it strengthens the theory of infection through the portal of perforation and detracts from the theory of sensitization to one's own pigment as a cause of sympathetic ophthalmia. I am aware that sympathetic ophthalmia without perforation, particularly in cases of tumor, has been reported, but it is so rare that the burden of proof is on those who report such cases.

Since many of the eyes showed detached retina and extensive hemorrhage in the posterior segment, I wonder whether the authors can venture an explanation as to why the incidence of fibrovascular membrane on the iris is so low. I believe it was mentioned in only 1 case.

I wonder whether Dr. Rones would care to say anything about the incidence of the Vossius ring, which Dr. Trygve Gundersen described so well as following contusion injuries in young people, and which does not occur in elderly persons. It is a pigmentary disturbance, with a diameter a little smaller than that of the pupil, and probably lies under the epithelium of the lens; it seems that this series would afford a wonderful opportunity to study the condition.

Regarding case 18, one of gunpowder injury, showing a rather extensive inflammatory response in the eye, I wonder whether this reaction could be explained on the basis of injury to the cornea. Studying the aqueous in rabbits, I found the response in cells and protein in the aqueous conspicuous after inflammation localized in the center of the cornea. For example, I found that it produced a much greater response, as indicated by cells and protein in the aqueous, than trauma of the same severity in the periphery of the cornea. If the cornea in this case had been extensively damaged by gunpowder, I should

expect an intense inflammatory response in the interior of the eye. No mention of the condition of the anterior surface of the cornea was made.

Finally, after reading this paper, one considers what benefit the physician called on to treat such injuries can derive from the pathologic studies Dr. Rones and Mrs. Wilder have presented.

Since hemorrhage and secondary glaucoma are the immediate complications, treatment should be directed against them. Such treatment might include complete immobilization of the eyes by snug double eye bandages, rest in bed, use of ice compresses, instillation of miotics and early surgical intervention for removal of blood. It would seem that wide incision and frequent irrigations of the anterior chamber, and possibly corneoscleral trephining, are necessary to accomplish this. The question of advisability of removal of a dislocated lens is to be considered. The use of calcium, a vitamin K preparation or any other means of inhibiting bleeding would seem important.

I wish to congratulate Dr. Rones and Mrs. Wilder on their present study of the material at the Army Institute of Pathology. We all await the results of their further analysis of the abundant material available to them.

Dr. Benjamin Rones, Washington, D. C.: We are deeply grateful to Professor Riddell and Dr. Irvine for their discussions of this paper. It is rather formidable to have thrown into your lap a paper of this scope. The reading of it was much simpler than the task the discussers had in wading through forty-seven detailed protocols and summaries of all the other material. We appreciate their attempt to make clinical sense out of this paper.

We also appreciate the audience's attention in the presentation of this material.

With regard to Professor Riddell's discussion, I again wish to emphasize that there were no cases in this series of penetrating injuries of the eye. The wounds are all nonperforating; consequently, no eyes in this series contained glass, metal or inorganic substances. There are many other eyes at the Army Institute of Pathology with penetrating wounds, and they will be classified and studied at some later date.

I am pleased with Professor Riddell's suggestion of the time limitation. However, he must bear in mind that a considerable number of these eyes were removed at field stations and hospitals where the operating surgeons were under great pressure. There was no follow-up study of the patients. After considerable difficulty, we arrived at the time-spacing relation on the basis of information that was as accurate as we could get, but not so detailed as a day to day notation would signify.

The value of this material lies in the fact that one has an almost experimental set of circumstances. If one is working with animals, one produces an injury, removes the eye in one, two or three days, or after any length of time desired, and then studies the damage and reparative processes that are present. For the first time we have this set of circumstances in a large series of human eyes, and I think therein is the chief value of this material.

Dr. Irvine asks: How can one tell when an eye will have recurrent hemorrhages? Pathologically, one can say that granulation tissue is composed of fresh young blood vessels; consequently, such capillary buds are likely, under many conditions of strain and stress, to give way and produce another hemorrhage in the eye. How soon blood staining of the cornea may occur it is difficult to say, because that depends a great deal on the tension of the eye and on how many pigment granules are forced to migrate into the corneal stroma.

With regard to the cases of early glaucoma, the earliest occurrence we had in this series was six weeks after injury. There are a number of cases of other types of injury in which cupping occurred considerably earlier.

With regard to the fibrovascular membranes, we mentioned their occurrence in 5 cases; and in many others in which they were not mentioned, organizing masses of hemorrhage in the anterior chamber and inflammatory exudates in the pupillary region and over the iris were observed.

Anatomic Variations in Attachment of the Oblique Muscles of the Eyeball. Dr. Walter H. Fink, Minneapolis.

An anatomic study was made of the superior and inferior oblique muscles. The problem was confined to the insertion of these muscles and their relationship to the adjacent rectus muscles. The work was approached from the clinical standpoint, and with the hope that, with a broader knowledge of the anatomic relations of these muscles, ability of ophthalmologists to diagnose and treat the obscure muscular defects would be increased.

Although the series of specimens was small and the data were restricted to the insertion of these muscles, certain conclusions were reached.

1. The anatomic variations in the insertion of the superior oblique muscle are more numerous that those of any other extraocular muscle. Variations in the insertion of the inferior oblique muscle are next in frequency.

2. The wide variation in the position of insertion of these muscles suggests the possibility that a "paretic" muscle may be abnormal in action because of an unusual insertion. It is likely that many of the obscure defects in the action of these muscles may be explained by a variation in the insertion into the eyeball.

3. The work indicates that a favorable and feasible surgical approach could be made at the insertion of the superior oblique muscle.

4. The study suggests the possibility of a more satisfactory surgical management of paralyses of the inferior oblique. Surgical intervention can, and should, be directed to the insertion rather than to the origin because of the easier approach to and the anatomic relations of the former.

5. The possible intimate relation of the inferior oblique muscle to the optic nerve and other important structures should be kept in mind in operations on the muscle.

Extraocular muscle surgery would take a step forward if a more accurate technic could be applied to procedures on the oblique muscles.

As matters stand now, possibilities for correction of a defect of an oblique muscle are limited. This state of affairs is due apparently to lack of knowledge concerning the anatomy of the oblique muscles.

It is surprising how few data are available concerning the variations in these muscles. The inquiring student is faced only with generalities. If the need for more exact data on this subject could be appreciated, more work would be forthcoming, and greater possibilities for scientific work on these obscure and poorly understood muscles would be opened.

DISCUSSION

DR. F. BRUCE FRALICK, Ann Arbor, Mich.: Dr. Fink has started on a commendable research project, which, when completed, will add materially to general knowledge of the anatomy of the extraocular muscles. The statistical analysis of the results of the investigation can be fully appreciated only after careful study of his beautiful display in the scientific exhibit. Only then is one able to comprehend the thorough and paistaking work needed to supply the information which he has given us today.

A fearful approach to muscle surgery is engendered by uncertainty regarding the diagnosis, physiology or anatomy of the extraocular muscles. Acquisition of knowledge of the anatomic relations presented by Dr. Fink will lead to better operations on these muscles than are now generally done.

The various measurements of the ocular muscles need not all be remembered, since not every one is of great clinical value. Some of the measurements given aid the ophthalmic surgeon in locating the muscles at operation. For instance, if the surgeon did not know that the global insertion of the inferior oblique muscle was about 10 mm. posterior to the insertion of the external rectus muscle, difficulty might be experienced in finding the insertion when recession or resection of this muscle is attempted. If it is remembered that the anterior edge of the superior oblique tendon is only about 2.5 mm. behind the nasal edge of the insertion of the superior rectus muscle, no trouble should be experienced in finding this tendon for resection or recession. Not all these data are available in any standard text of ophthalmology or ophthalmic anatomy.

For many years my colleagues and I have been teaching our residents the necessity of retaining normal anatomic relationships of the extraocular muscles when performing operations on these muscles. The surgeon should have great respect for the fibrous sheath of the muscle and leave it intact. Stripping of the sheath from the muscle and tendon leads to adhesions of the muscle to the sclera.

Dr. Fink has emphasized that in performing recessions not only should the tendon be spread out to the normal breadth of insertion present in the particular eye, but the recession should be measured equally from the two ends of the scleral insertion. The only exception to this rule is in those rare eyes in which the line of insertion is definitely abnormal. A muscle cannot be reattached to the globe with a single central suture with the expectation that that attachment will have a spread of approximately 10 mm. Very likely a narrow point of

attachment would have little effect on the efficiency of the lateral and medial rectus muscles when the eye is moved in the horizontal plane.

This narrow point of insertion could conceivably have a serious effect on the efficiency of these muscles when they are used in the oblique or the vertical position of gaze.

I can see no excuse for the rather common practice of reinserting a medial or a lateral rectus muscle higher or lower in an attempt to correct a hyperphoria or hypertropia. Such a practice is an admission of inadequate diagnosis of the anomaly at hand.

I agree with Dr. Fink on the advisability of spreading the scleral insertions of the oblique muscles to their normal width when recession or resection is done. This principle should apply as much to the oblique muscles as to the rectus muscles. It is far more difficult to reattach the oblique muscles than the rectus muscles, and considerably more manual dexterity is required to insert two sutures in order to obtain the normal width of the attachment. This is especially true when recession of the inferior oblique alone is performed, without resection of the external rectus. More exposure is afforded when recession of the inferior oblique is combined with resection of the lateral rectus, but even then I have found difficulty in placing the posterior suture far enough back on the globe.

My poorest results from recession of the inferior oblique have been due to incomplete severance of the scleral attachment posteriorly. Since this attachment extends within 5 mm. of the optic nerve, one is working blindly with scissors in making this section. It is, therefore, not difficult to leave some of the posterior fibers intact.

I cannot agree with Dr. Fink's interpretation of the line of action of the inferior oblique and his method of suturing the inferior oblique to the sclera along this line. If he reattaches the anterior end of the tendon at the same distance from the cornea as it was originally he is placing the new insertion too far posteriorly and his new line of action will be more oblique than formerly. It is my belief that the new insertion should be along the normal line of action of the muscle. This line of action extends obliquely forward and down around the globe.

Dr. Fink has pointed out the relation of the superior rectus to the superior oblique. I feel that an even more important relationship is that of the superior rectus and the levator oculi muscle. The union of the superior rectus and the levator muscle is so strong that if the amount of recession of the superior rectus is too great the upper lid will be elevated, giving an apparent exophthalmos on this side. On the other hand, if the superior rectus is resected too much, relative ptosis will result. The same relationships are present in the lower lid, although here there is no levator tendon. Fascial bands passing from the inferior rectus to the lower tarsus act in the same manner as the levator muscle of the upper lid. Too generous recession of the inferior rectus widens the palpebral fissure as a result of the lower lid being pulled down. Too extensive resection of the inferior rectus causes elevation of the lower lid, with resultant narrowing of the palpebral fissure.

DR. RAYNOLD N. BERKE, Hackensack, N. J.: Dr. Fink's presentation is timely and to the point. It is timely because never before have so many operations been proposed for the treatment of hypertropia

associated with overaction or underaction of the oblique muscles. These operations may be briefly noted as follows:

(*a*) To decrease the action of the inferior oblique muscle, Dr. J. H. Dunnington, a number of years ago, advocated myectomy, tenotomy or myotomy of the overactive muscle. Later, Dr. J. W. White described an operation for recession of the inferior oblique muscle to decrease its action.

(*b*) To increase the action of the inferior oblique muscle, Dr. J. M. Wheeler designed an operation for advancing the tendon of this muscle over the lower margin of the orbit. Later, Dr. J. W. White suggested tucking the inferior oblique muscle to increase its action.

(*c*) To increase the lifting power of the superior oblique muscle, Dr. Wheeler, a number of years ago, designed an operation for tucking the reflected tendon of this muscle. Recently, Dr. J. M. McGuire gave a description of an operation, which I understand is now in print, for resecting the tendon of the superior oblique muscle to increase its action.

(*d*) To decrease the action of the superior oblique muscle, Dr. W. L. Hughes advocated recession of its pulley. Dr. Fink suggested that it may be feasible to tenotomize the superior oblique muscle lateral to or under the superior rectus muscle. Three months ago I described an operation for tenotomy of the superior oblique on the nasal side of the superior rectus.

Thus, a total of eight operations are available for the treatment of hypertropia associated with dysfunction of one or more oblique muscles.

Surgeons who perform these operations frequently say that they are easy and simple to do. Surgeons who do them rarely, or not at all, believe that they are difficult. This difference of opinion, I believe, is due to familiarity with the anatomy of those who do the operations frequently.

Dr. Fink's paper brings up a number of interesting and practical points. I should like to enlarge briefly on two or three of them. He has shown that the insertion of the inferior oblique muscle begins about 10 mm. posterior to the inferior edge of the insertion of the external rectus muscle and from this point recedes backward in the average case for a distance of about 10 mm. The entire insertion of the inferior oblique usually lies under the inferior half of the belly of the external rectus. Obviously in doing a recession of the inferior oblique muscle, it is necessary to keep this relationship in mind. One should also remember this relation in every operation on the external rectus, for it is possible accidentally to tenotomize this muscle during a resection, recession, tucking or advancement of the muscle. I know of such an accident, in which the inferior oblique muscle was accidentally tenotomized during a routine resection of the external rectus muscle [drawing explaining how accident may develop]. After the conjunctiva has been incised and an opening made in Tenon's capsule, the muscle hook is passed under the external rectus muscle, and during this maneuver the belly of the inferior oblique may be accidentally picked up, because the insertion is farther forward than normal, because the hook is passed too deep into Tenon's space or, for some other reason, the inferior oblique muscle is brought forward to a position directly under the insertion of the external rectus muscle. One can readily see how easy it would be for the surgeon, when freeing the external

rectus muscle from Tenon's capsule, to sever accidentally the inferior oblique muscle. In the case to which I referred, a second operation was done, at which time the inferior oblique was observed to be attached to the sclera at the insertion of the external rectus muscle, having been completely severed from its normal attachment. It is necessary only to keep this possibility in mind in operations on the external rectus muscle to avoid this complication.

Dr. Fink has shown that the reflected tendon of the superior oblique muscle passes only 2 or 3 mm. posterior to the insertion of the superior rectus muscle. This is important, as already brought out by him and Dr. Fralick, because in doing an operation on the superior rectus muscle the tendon of the superior oblique muscle may accidentally be severed. I know of one such accident.

Dr. Fink has suggested that it may be possible to tenotomize the superior oblique muscle lateral to the superior rectus muscle. In this he is correct, but, unfortunately, tenotomy of the superior oblique muscle at this place is difficult and uncertain. It is difficult because the insertion of this muscle extends so far posterior to the equator. It is uncertain in its effect because one cannot be sure that all the fibers have been severed from the globe. Unless they are all severed, little or no effect will result. Of 4 tenotomies of the superior oblique done lateral to the superior rectus at the Institute of Ophthalmology of the Presbyterian Hospital, only 1 was entirely successful. In the 3 unsuccessful operations, the failure was attributed to incomplete tenotomy.

A better site of tenotomy of the superior oblique muscle is on the nasal side of the superior rectus muscle. This is the site of choice, for the following reasons:

First, the reflected tendon of the superior oblique muscle at this point is well forward, being only 2 or 3 mm. from the insertion of the superior rectus muscle.

Second, the width of the tendon of the superior oblique muscle at this point is only 5 or 6 mm., the tendon being compact. These two features make it easy to pick up the reflected tendon of the superior oblique muscle.

Third, if the operation is done at this point, it is not necessary to remove the superior rectus or to disturb its insertion, as would be the case if the operation were done lateral to the superior rectus muscle.

Fourth, there are at this point no important nerves, arteries, veins or other periocular structures which might accidentally be injured during the operation.

Finally, if the operation is done here, it will be done under Tenon's capsule and within the sheath of the superior oblique muscle. If the operation is done here, complete paralysis of the superior oblique muscle will not follow the operation.

Dr. Walter H. Fink, Minneapolis: In answer to the question of interpretation of the anatomic variations: I cannot with certainty diagnose a case of abnormal insertion. There is the possibility that in a certain percentage of cases the abnormal muscular action is due to an abnormal insertion.

I believe that tenotomy of the superior oblique should be done on the tendon, and not on the insertion. I think that Dr. Berke is correct, and I am in full agreement with his approach, for to me it is the most logical procedure.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Brittain F. Payne, M.D., *Chairman*

Milton L. Berliner, M.D., *Secretary*

April 21, 1947

INSTRUCTION HOUR

Fitting of Glasses for Aphakic Eyes. Dr. Daniel B. Kirby.

Condition of the Eye After Cataract Extraction. Dr. Daniel B. Kirby.

PAPERS OF THE EVENING

New-Formed Vessels in the Fundus Oculi. Prof. A. J. Ballantyne (by invitation), Glasgow, Scotland.

The terms "new-formed" and "new-built" as applied to certain vascular anomalies of the fundi are not strictly accurate but are convenient expressions to describe vessels which make their appearance under pathologic conditions in the vascular tissues of the fundus or grow from such tissues into a normally avascular neighborhood. They vary greatly in form and may be intraretinal, subretinal or preretinal. They can be studied with the ophthalmoscope in the living eye, with the loupe or the binocular microscope in the excised eye and with higher magnification under the microscope, in bulk or in sections. Nothing quite analogous is known in general pathology. Such vessels may exist but are not recognizable in the course of ordinary histologic examination.

The most familiar forms of new vessels are seen in the networks of venules spread out within, or on the anterior surface of, the retina, the so-called rete mirabile, and, often associated with these are tufts, or glomerular formations, of fine veins projecting into the vitreous, embedded in an almost invisible supporting tissue. Most, if not all, of these intraretinal networks can be formed by the expansion of preexisting venules and capillaries. They are usually encountered in cases of obstruction of retinal veins and provide a collateral circuit to accommodate the venous blood stream.

The preretinal vessels and vitreous tufts occur in similar circumstances and may have a similar function, but their purpose is not understood. It has been suggested that the vitreous tufts perform the function of removing retinal exudates and hemorrhages. The preretinal vessels are correctly described as newly formed; they originate from the retinal veins by budding. They are able to penetrate the internal limiting membrane and are not necessarily preceded by retinal hemorrhage.

Varicose vessels on the optic disk also occur when there is venous obstruction, usually thrombosis of the central retinal vein or one of its branches. They also provide a by-pass for the venous circulation. They may be formed by the expansion of preexistent vessels on the disk. Artery-to-vein and artery-to-artery anastomosis are also, but less frequently, observed in the retina, and an unusual form is anastomosis of a retinal vein with the choroidal vascular system through a small choroidal coloboma.

The vessels which originate in the choroid, perforate the membrane of Bruch and spread between the choroid and the retina as a vascular membrane (Reichling and Klemens) are seen ophthalmoscopically in association with senile central exudative and hemorrhagic retinitis and may be described as one of the forms of angioid streaks. These vessels, like the preretinal networks, may have the function of carrying off the harmful products of local disease.

Histologically, most of the new-formed vessels have the appearance of greatly enlarged capillaries, but there is a type of much larger new-formed veins, seen chiefly in diabetes, with walls of varying thickness, which present the characters of phlebitis, phlebosclerosis and hyaline and fatty degeneration.

An interesting type of new-formed vessels, which so far has been identified only histologically, has been described by Prof. A. Loewenstein (1946) as intramural vessels. They occur in cases of vascular disease, are situated within the walls of the vessels, communicate here and there with the lumen of the parent vessel and sometimes form an elongated network in the vascular wall.

The line of demarcation of these vessels from the vessels which canalize a venous thrombus, or which appear in the optic nerve as collaterals of the obstructed vein, has not been determined. They have certain points of resemblance to the intramural vessels described by Winternitz and associates (The Biology of Arteriosclerosis, Springfield, Ill., Charles C Thomas, Publisher, 1938) as occurring in the aorta, the renal arteries and other large vessels.

DISCUSSION

DR. BERNARD SAMUELS: I am not prepared to discuss Professor Ballantyne's fine presentation, but I should like to ask him for the anatomic reasons which brought about the secondary glaucoma and loss of the eye in his case. In some cases of occlusion of the central vein glaucoma develops and necessitates the loss of the eyeball, whereas in others the condition goes on for years and glaucoma does not develop.

PROF. A. J. BALLANTYNE, Glasgow, Scotland: I am sorry that I cannot throw light on this unsolved problem. I have no personal knowledge of that point, and I should not like to speculate on it.

DR. ARNOLD KNAPP: It is a great privilege to hear Professor Ballantyne speak on a subject to which he has made such important contributions—a subject which is fascinating and, at the same time, very difficult. I am particularly impressed by his demonstration of the formation of new nessels on the head of the optic nerve and of the development of new vessels which, after they break through the inner layer of the retina, spread out under the hyaloid membrane. I should like to have him give us his interpretation of the reasons for the tremendous development of new vessels in cases of what is regarded as malignant diabetic retinitis.

PROF. A. J. BALLANTYNE, Glasgow, Scotland: My interest in these new-formed vessels really began in the study of diabetes. In cases of diabetic retinopathy these gross changes appeared in the retinal vessels, and it was only in cases of diabetes that Dr. Loewenstein and I were able to demonstrate the perforation of the retina. There was only 1 exception to this in the material which we examined, the case being

the one first illustrated on the screen, with a peculiar plexus in the course of the superior temporal vein. The case was one of hypertension without glycosuria. The state of the blood sugar was not recorded. The sections showed well the vein breaking through the anterior layer of the retina and forming a plexus which spread over the surface. I cannot help feeling, in spite of that exception, that there is a specific connection between diabetes and these gross changes in the retinal vessels. Comparison of a case of diabetic retinopathy and one of hypertension with vascular changes in the retina shows a difference in the history of the retinopathy, as well as in the ultimate changes in the retina, in the two conditions. In the case of diabetes, there is no discoverable connection between the disability or the age of the patient, or many other factors, and the presence or severity of the retinopathy. A patient with diabetes may never show more than fulness of the retinal veins. Again, a patient may show nothing more than minute capillary networks and aneurysms in the retina, and these may remain unchanged for years. We have had 1 or 2 patients under observation for four or five years without alteration in these lesions. Then, a sudden change for the worse may occur in the course of the diabetic retinopathy, with the appearance of gross vascular lesions, hemorrhages, exudates and changes which destroy the eye, even though the patient is comfortable and the diabetes is well controlled. In cases of hypertension the first sign is the presence of minute hemorrhages radiating from the disk. At the same time woolly patches and other exudates appear. Sooner or later retinal changes not found with diabetes occur, chiefly the result of arteriosclerosis, and it is only at a late stage that evidence of thrombosis of the retinal vein is observed and a network of new-formed vessels is seen. Another fact of importance is that by the time the hypertensive patient has manifested advanced changes of this kind his expectation of life is short. Except for persons with venous thrombosis and secondary glaucoma, one does not often see a patient with hypertension living for years with blindness due to retinopathy. But in the case of diabetes a comparatively young patient may lead a reasonably healthy life but be blind, owing to the presence of exudates and hemorrhages. These two conditions are in distinct contrast, and it is our feeling that the combination of hypertension and diabetes does not have much to do with it, but that there is something specifically different in the two conditions which makes them develop along different lines. At least 50 per cent of our patients with diabetic retinopathy do not have hypertension.

DR. JOSEPH IGERSHEIMER: Professor Ballantyne has demonstrated an interesting case of thrombosis of the central vein without retinal hemorrhages. I should like to ask whether the diagnosis in this case was made clinically, or only microscopically, and, second, whether the glaucoma, as usual, followed the thrombosis, or whether it came first and the thrombosis later?

PROF. A. J. BALLANTYNE, Glasgow, Scotland: For several years the case was diagnosed as one of thrombosis on the basis of the appearance of the retinal veins and those on the disk. The condition was anomalous in the absence of hemorrhage. I cannot tell at what stage the diagnosis of glaucoma was made.

Medical Needs of the Philippine Islands. Dr. Hermenio Velarde (by invitation), Manila, Philippines.

Dr. Velarde spoke interestingly on the conditions during the siege of Manila and of the plight of ophthalmology, as well as of medicine in general, in the Philippines during the reconstruction.

DISCUSSION

Dr. Brittain Ford Payne: My first visit to one of the war-wrecked medical centers of the Philippine Islands was early in May 1945. All that remained standing and serviceable was a portion of the Philippine General Hospital, which is about a mile from the city wall of Manila.

All that was left of the recently completed clinic and laboratory buildings was charred walls and mangled equipment. Live "booby traps" or bombs were concealed in the rubbish and ashes. The administration building of the hospital was so damaged by shells and bombs that it was abandoned. A strafed ambulance stood in front of the building as a reminder of the ferocious battle for Manila two months before.

Other buildings were so damaged by the retreating Japanese that they will have to be rebuilt from the ground up. The medical school, the cancer institute, the school of nursing and the postgraduate school, all modern and well equipped buildings, were completely gutted and wrecked.

With so much of the physical plant ruined by battle, and so much suffering from wounds, starvation and death, the civilian doctors and nurses of the staff stood by to render what aid they could. The civilian doctors worked night and day with the sick and wounded, native and American alike, not knowing whether the next shell or bomb would strike them. The day after the Japanese were pushed into the walled city, the hospital was operating with every available cot or bed taken by sick or wounded. Many single beds were occupied by mother and child, both sick or wounded, and some were lying on the bare floor.

An inspection of the main hospital showed that some additional space was available, though there were craters in the floor and roof and parts of the walls were gone. Immediate repairs were made, and hundreds of patients were given shelter. Practically all laboratory equipment was gone; x-ray machines were destroyed, and the library was burned to the ground. Only three microscopes could be salvaged for the entire center, and these had been hidden away in the early days of the Japanese occupation.

It is difficult for us to visualize the hopelessness of the situation as it was in Manila during the Japanese occupation and while liberation was in progress. Most of the doctors in this country would find it difficult to work with bare hands, no drugs and little or no equipment. The Filipino doctors found it difficult also; but they had had some experience under Japanese domination, and they are doing their best today with the little they have. The American doctors, for example, Musgrave, Strong and Heiser, would be proud, if they knew, of the excellent record made by the great medical center they founded and the physicians they trained.

The University of the Philippines was founded at the turn of the century by Americans, and the medical center was a major part of the

university. The other great center and medical school in Manila was a part of the University of Santo Tomas. It was used by the Japanese as a crowded prison camp and by the American Army as a military hospital after the liberation. It will be reopened for classes as soon as its war damage is repaired and new buildings can be constructed. Both these fine institutions need immediate aid in the form of books, instruments, drugs and magazines. The libraries have been pilfered and burned, and hundreds of Filipino doctors have not seen a copy of *The Journal of the American Medical Association* since December 1941. This is true of other journals and books. All reference copies have been lost, and old, as well as new, journals are needed for teaching and investigation. Instruments for surgery and clinical investigation are badly needed. American ophthalmologists have been most liberal in furnishing instruments, but the need extends to equipment for general surgery and medicines as well.

Equipment for the study and drugs for the treatment of tropical diseases should be sent to Manila as soon as possible. It has been wisely said by Dr. Velarde that our progress in treatment of tropical diseases in this country will be advanced in proportion to our interest in research in the Philippine Islands.

The program of research on tropical diseases would be stimulated by our sending literature and material for investigation. Many Filipino physicians come to our great medical centers for training, and many of them are graduates of our medical schools. The dean of the medical school, Dr. A. G. Sison, is an alumnus of the University of Pennsylvania and has kept standards well within the requirements of the Association of American Medical Colleges. The Philippine Medical Association is similar to the American Medical Association. Interchange of students and research workers should be encouraged in order that we may understand tropical disease and treat many of our veterans so afflicted.

We Americans should never forget the aid the Filipinos gave us while so many of our citizens were imprisoned by the Japanese. We should never forget the aid loyal Filipinos gave MacArthur's army, and we should help them in reestablishing themselves medically and economically. They do not need money as such, but they do need books, instruments and drugs.

An Integrated Artificial Eye and "Vitallium" Implant for Use in Enucleation and Evisceration. Dr. Wendell L. Hughes, Hempstead, N. Y.

Three types of implants have been designed, all in "vitallium," each of them to be integrated with the same type of artificial eye so that the motion of the implant is imparted to the eye and the eye is held up to support the upper lid in a normal manner.

All the implants have the same type of face, which is left exposed in the socket, with an oval depression anteriorly. The eye is designed with an oval peg on its posterior (concave) surface to fit into this depression.

1. *Enucleation Implant.*—The first type, for use at the time of enucleation, has a circular attachment bar, to which the rectus muscles

are attached in the horizontal and vertical positions and the fascia bulbi is attached in the quadrants of the four oblique muscles.

2. *Replacement Implant.*—The second type has a smaller body posteriorly and is designed to replace the usual type of buried implant now in use. This implant has a similar ring for the attachment of the muscles and fascia.

3. *Evisceration Implant.*—The third type, designed for use at the time of evisceration, has a similar anterior face and fits inside the sclera. The evisceration is performed and the implant is inserted through an incision posterior to the insertion of one of the rectus muscles. The muscle tendon is first detached and is replaced later onto the anterior lip of the scleral wound to aid in holding it together. The cornea is removed to allow the anterior surface of the implant to protrude through. Movement of the eye is better than with the conventional type of implant because the motion of the implant is transferred to the eye by means of the integrating depression on the implant and the peg on the posterior surface of the eye. In the old, buried, type of implant there must of necessity be considerable lost motion or slippage between the implant and the eye.

The upper lid is supported because the eye is held up by its direct connection with the implant, eliminating the depression usually present with the conventional type of artificial eye, the entire weight of which must rest entirely on the lower lid.

Kodachrome slides and moving pictures illustrated the details of design and technical procedure in use of the replacement implant.